THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—32ND YEAR. SYDNEY, SATURDAY, FEBRUARY 3, 1945. No. 5.

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FAMILIAL CORTICAL CEREBELLAR ATROPHY: A CONTRIBUTION TO THE STUDY OF HEREDO-FAMILIAL CEREBELLAR DISEASE IN AUSTRALIA.

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AUSTRALIAN medical literature contains few articles upon the subject of cerebellar disease. In 1941 we published in *Brain* a report of a family, resident in Australia, suffering from a type of cerebellar atrophy. A description of those members of the family who had been studied was given and the pathological and histological details of a case in which autopsy had been performed were included.

Another member of the family has died, and an opportunity has been given us of examining, for the second time, the structures affected. As this family is now scattered widely throughout this continent, we have considered it advisable to publish again a description of the family with the clinico-pathological picture presented by those affected. With the growth of the family further examples will almost certainly be seen.

It is appropriate that this laboratory should be associated with the first description of these cases in an Australian journal, since Campbell and Litchfield's case of Friedreich's ataxia, the first in Australian literature, was also worked up here.

In this report a brief *résumé* of the family history will be given, and a clinical record of the member whose brain is under review, with morbid anatomical and histological detail, will be presented. A synopsis of the family tree is given for convenience of description. A circle indicates members of the family known to have been affected up to June, 1940. A cross indicates those affected and examined by us.

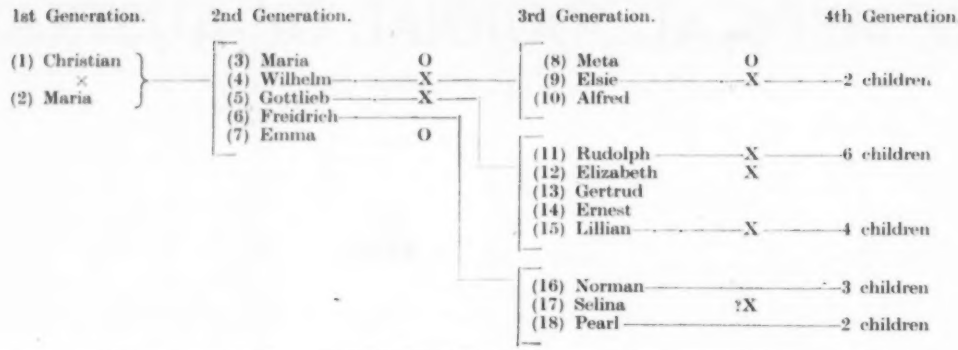
About seventy-five years ago a young German settler named N. (who, it is believed, came from near Leipzig) arrived in Australia with his wife and several young children. Very little is known of the parents. The husband (number 1) was "never very strong", suffered from asthma and died at the age of forty years in 1873 from some unknown cause. His wife (number 2) is reputed to have been a very strong and healthy woman up to the time of her death at the age of eighty years.

In the next generation, number 6 (Freidrich) died at the age of forty years as a result of "blood poisoning". All other members of that generation were affected by this disease. Wilhelm (number 4) was seen by us prior to his death in 1938, at the age of eighty years. We were subsequently able to perform an autopsy, and the report of his case has already been published.[1] Gottlieb (number 5) was examined in 1939 in Queensland by one of us (B.H.), and a history and description of him as he appeared at that time are given below. He died in 1942, aged eighty-two years, and a report on the examination of his brain is included here also.

In the third generation, number 10 died an accidental death at the age of twenty-seven years, and number 4 died at the age of nine years of "sunstroke". Numbers 8, 9, 11, 12 and 15 all suffer from the disease, and, with the exception of number 8, have all been examined by one or more of us. Another member of this generation, number 17, was "suspect" in 1940, but has not been seen by us since.

Most members of the fourth generation have been examined. In 1940 their ages ranged from eighteen years to infancy. One boy, a son of number 11, was in 1939, for various reasons, considered likely to develop the family disease, but all the other children appeared quite normal. We have had no opportunity, owing to the war, of reexamining those members of the family suspected of developing the complaint.

A branch of the family, apparently descended from a member of the second generation, is known to exist in South Australia. Previously we were unable to trace this



branch, but in 1942 one of us (B.H.) had under his care in Tripoli (Lebanon), a soldier who was a member of the South Australian family, and inquiry of that soldier indicated that several members have also suffered from this disease.

Thus, although this family is not as yet a large one, the disease is known to exist in New South Wales, Queensland and South Australia.

Clinical Features.

Seven members of the family suffering from this disease have been examined.

It is not possible to make any definite statement regarding the age of onset of this malady. In the second generation the age of onset has been in the middle fifties; in the third generation the average age of onset is approximately forty years. It seems not unlikely that instances will be seen at even earlier ages in subsequent generations if the "anticipation", usually found in familial disease, takes place.

The disease appears to have had no appreciable effect on longevity. In the second generation the ages at death of the affected members ranged from sixty to eighty-two years. In the third generation, number 8, who is aged fifty-four years, is now said to be bedridden; but the other affected members of this generation are all at least "able to get about", and their ages range from the middle of the fifth to the middle of the sixth decades.

The initial symptoms have always been a disturbance of gait or a disturbance of speech. In three of the seven cases examined by us a disturbance of gait was the first symptom; in two, disturbances of speech and gait appeared at about the same time. In addition to the earlier involvement of the lower limbs, they are affected more severely than the upper. The ataxia is typically cerebellar.

Disturbance of gait, like other manifestations of the ataxia, is insidious in its onset, and so slowly progressive that signs of the disease have usually been well established before the sufferer is more than vaguely conscious that there is "something wrong". Indeed, the presence of neurological disease has escaped the notice of at least one medical practitioner in every case we have seen.

With the progress of the disease, symptoms of cerebellar disorder increase. Muscular hypotonia has not been a feature in these cases; even in advanced stages of the disease, muscle tonus has usually been normal. The tendon reflexes do not disappear as a rule.

Nystagmus probably develops in all instances sooner or later, but on available evidence it does not appear to be one of the earlier manifestations.

The dysarthria is somewhat distinctive. It has varied between the extremes of a slight slurring of words and a tendency to run syllables and words into one another in the earlier stages, up to unintelligibility in the most advanced stages. In all, however, there has been a common quality of voice and speech difficult to describe, but none the less distinctive. This may be better conveyed in the description of number 5 below.

Difficulty in swallowing was evident in three of our cases; it is doubtless due to an ataxia of the normal mechanism. In no instance was it a serious disability.

Athetosis accompanied the other neurological disturbances in one case (number 4), and in another (number 11) there was a suggestion of Parkinsonism. It is not suggested that these extrapyramidal phenomena are an integral part of the disease; we are merely reminded of the ramifications of the cerebellar connexions.

It is interesting to note that in the early stages in the majority of these cases the diagnosis of "joint disease" had been made.

Clinical Record of Number 5 (Gottlieb N.).

Gottlieb (number 5) was examined by one of us in southern Queensland in 1939, at the age of seventy-nine years. He died at the age of eighty-two years. The report is given in some detail as it was published previously in *Brain*, for it illustrates the slow development of the disease and the clinical picture of the fully developed condition.

> He was born in Germany and came to Australia as a child. As a young adult he commenced farming, and continued in this occupation all his active life. At about the age of 55 he fell from a horse, and although his injuries were not of a serious nature he was confined to his bed for a short period. When he recommenced his usual activities, he noticed some difficulty in walking, especially in the use of the left leg, which he described as a "stiffness of the left hip".
>
> During the ensuing five years he experienced increasing difficulty in walking, and because of this he was obliged to give up his work, though he could still "potter around the garden".
>
> Eighteen or nineteen years ago, through his disability, he fell against a bed and "strained a muscle in his leg". He was admitted to a country hospital, where he remained for a short period. (We have been unable to obtain any records from the hospital.) On leaving his bed he was unable to walk without help, and indeed has not walked without help since that time. In the earlier years the support of one person was sufficient to maintain him, but in the past two years he has required one person on either side, and for many months past he has had to be almost carried. He has been able to dress and feed himself and still does; but during the past seven years he has been handicapped in both respects "by a great shakiness". Dressing and meals occupy a great deal of his time as a consequence.
>
> His speech became affected at about the age of sixty. It was first noticed that he seemed to slur some words, or clip words, and ran syllables and words into one another. The deterioration of speech was slowly progressive. During the last five years his speech had been difficult to follow, and in the past year he had become almost unintelligible. He had been able to swallow apparently normally, but it had been observed during the past year or so that he frequently dribbled. He had had failing eyesight due to bilateral cataract, and he had had some difficulty with micturition due to enlarged prostate.
>
> He had pneumonia at the age of 17, and an attack of jaundice, associated with pain, at the age of 67.

Examination revealed a somewhat stout old man, with a mop of grey hair and a thick white beard. He spoke with an ataxia of speech characteristic of other affected members of his family, though in a more advanced degree. His voice was high-pitched and monotonous, and his speech was followed only with the greatest difficulty. Words were slurred and syllables and words ran into one another—for example, "Lefairsetemer" was his rendering of the statement "left there in September"; he described a headache as "Bacmied" ("back of my head"), and said that his family came from "Licey" (Leipzig). He was cheerful. He gripped one's hand firmly but shakily. He exhibited no athetosis.

FIGURE I.

Sagittal section through the vermis of "N" (right), compared with same section of the vermis of a male of similar age (left); cerebella vary in size, but the atrophied and pycnotic folia of the cerebellum of "N" in the regions of the culmen and clivus merit notice, as does also the thickened pia covering them. (Natural size.)

There was a moderate intention tremor in the upper limbs in picking up objects, but this, by contrast with the movements of his legs to be described, was slight. His intelligence was normal, and he cooperated satisfactorily.

He walked with the help of two grandsons, one on each side holding him firmly under each arm. As each

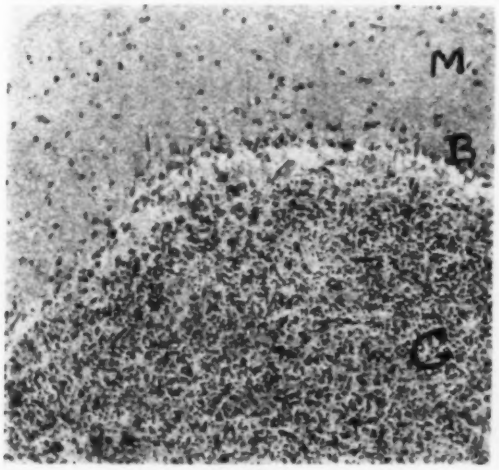


FIGURE II.

This section of a part of a cerebellar folium, typical of all the others, reveals no Purkinje cells, and the molecular layer M is separated from the granular layer G by a rarefied area bounded externally by a row of hypertrophied glial cells (the Bergmann glia) forming a palisade. (Hæmatoxylin-eosin stain, × 100.)

leg was advanced it was lifted from 6 to 8 inches from the ground and thrown outwards and forwards in a grossly ataxic manner, and planted flatly and firmly on the ground before the other foot was raised. He walked on a very wide base.

Cranial nerves: The fundi could not be seen on account of cataract formation. There was a rapid fine nystagmus when he looked to the left with the slower phase returning to the central fixation point. To the right the nystagmus was rather slower and coarser, again with the more rapid phase away from, the slower phase towards, the central point. The ocular movements were otherwise full and normal, and the functions of the other cranial nerves appeared undisturbed.

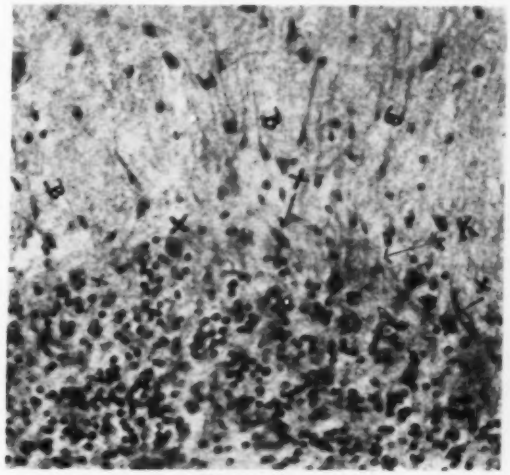


FIGURE III.

Similar section to Figure II. The Bergmann glia cells B, B are enlarged, point upwards, and produce visible perpendicular fibres; baskets are indicated at K, and sclerotic pycnotic cells, possibly remains of Purkinje cells, at X, X. (Iron hæmatoxylin and Van Gieson stains, × 200.)

Motor: There was no evidence of muscular wasting; power was remarkably good in the arms and the legs, and apparently good in the muscles of the trunk. There

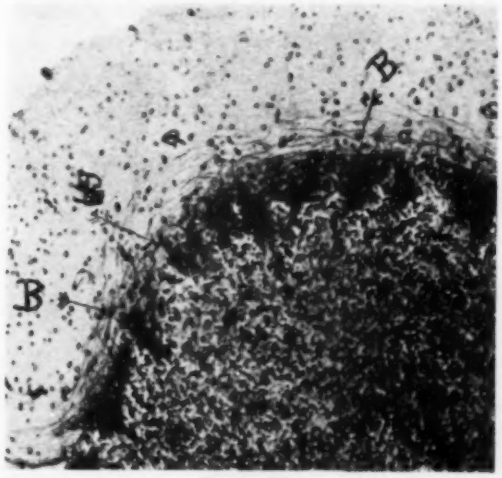


FIGURE IV.

This silver impregnation reveals the collapsed baskets B (actually in form a bundle of faggots), empty of Purkinje cells, the fibres being probably swollen. Horizontal fibres were seen at Q. (Cajal silver stain, × 150.)

was some ataxia of movement in the arms (e.g. handshake, picking up objects, etc.), but it was not of a severe grade. In putting the tip of the finger to the tip of the nose he hesitated only slightly before reaching the nose, shook slightly as he placed the finger on the nose, and slightly rubbed it in. This was a little more noticeable on the left side.

His legs were grossly ataxic in the execution of all movements. When asked to put a heel on the opposite knee and run the heel down the shin to the foot, the leg employed moved wildly in the air before settling on the knee, the heel thereafter following a jerky and erratic course down the front of the shin to the ankle.

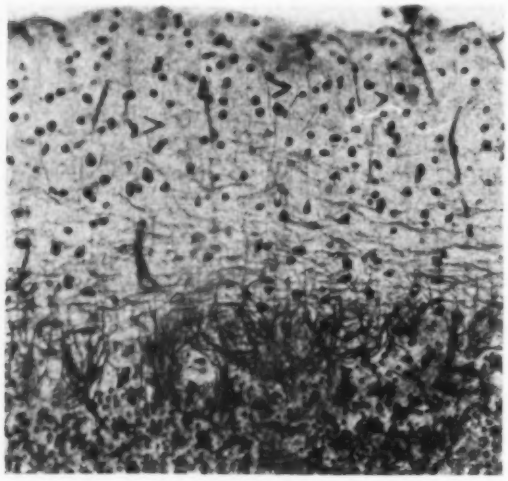


FIGURE V.

This thinner section allows one to see through the baskets; a few extremely dark, shapeless points may be degenerated Purkinje cells, they are smaller than the Golgi type II neurones. Vertical fibres may be seen ascending in the molecular layer (climbing fibres). The fate of the "tendril" fibres is not indicated. (Cajal silver stain, × 200.)

Attempts to touch the observer's finger with either great toe were also very ataxic, especially with the left limb.

Reflexes: The tendon jerks were all present and normal. The abdominal reflexes were elicited with difficulty on account of his obesity, and the cremasteric

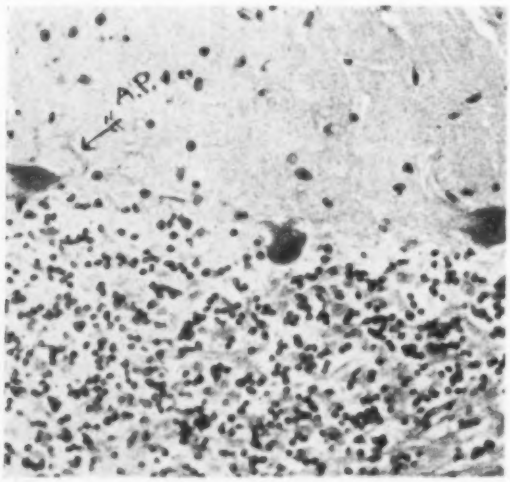


FIGURE VI.

This section from the central lobule shows three quite fair Purkinje cells with evidences of an antler at A.P. Perhaps five or six others equally as good were encountered in this section, and nowhere else in all sections examined. (Hæmatoxylin-eosin stain, × 400.)

reflexes were present. The plantar reflexes gave a plantar flexion response.

No evidence could be found of bone or joint disease, nor of any former fracture.

The size of the heart could not be determined clinically owing to the patient's obesity; the heart sounds were clear; brachial and radial arteries, so far as it was possible to determine, were normal. The blood pressure was 148 systolic and 96 diastolic.

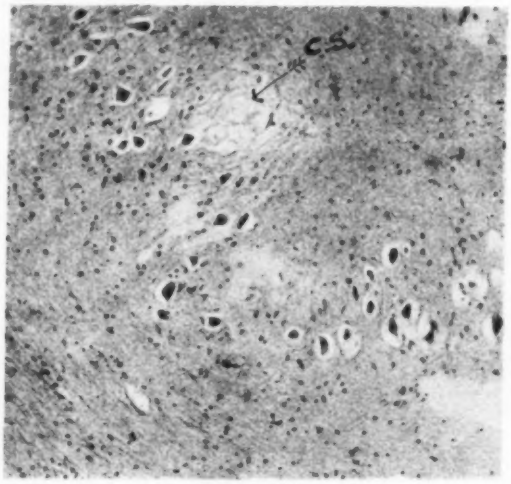


FIGURE VII.

The neurones of the dentate nucleus, though deeply pigmented and somewhat pycnotic, yet possessed good myelinated processes. Note the cyst-like spaces. Indications of the myelinated fibres around are seen on the left side at the bottom. (Hæmatoxylin-eosin stain, × 200).

Examination of all other systems revealed no abnormality.

The external genitalia were normal, as also the pubic hair.

Urine normal.

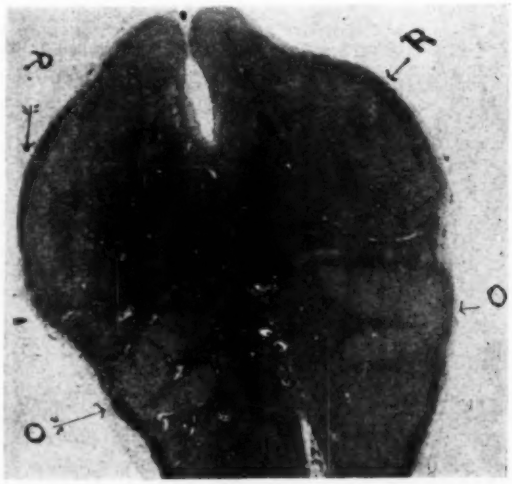


FIGURE VIII.

This oblique section of the upper part of the medulla reveals the olives O, O as too pale (diminution of olive myelinated fibres), too small and uneven. The other tracts stain well, including the restiform bodies R, R, which are unfortunately distorted because they were cut obliquely. (Weigert-Pal stain, × 4.)

Pathological Aspects.

The family history, clinical signs and pathological findings in the case of the brother being known, the similar clinical signs in the present case warranted a search for a similar pathological explanation. It was desirable, of course, to survey anatomical structures which might

indicate any other cerebellar abnormality, such as Thomas's olivo-pontine-cerebellar type of atrophy, Marie's or Friedreich's spinal cord involvement and the superior cerebellar peduncle types of atrophy.

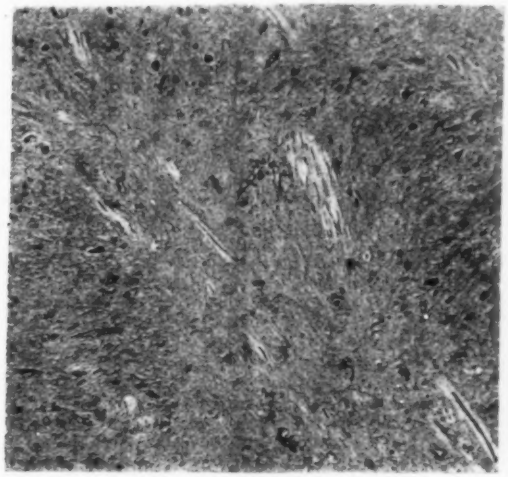


FIGURE IX.

Close-up view of portion of an olive. The few remaining olive neurones are picked out in blue (very dark), while the innumerable other structures are hypertrophied glia cells. (Koneff's stain, × 200.)

To the naked eye the brain seemed on the small side (the right cerebral hemisphere weighed approximately 440 grammes and the left hemisphere 475 grammes). The pia-arachnoid was fairly normal, and in the frontal pole rather more evident sulci were present than in the occipital pole. The cerebellum also was small (weighing 93 grammes). The cerebrum:pons-medulla:cerebellum ratio was thus

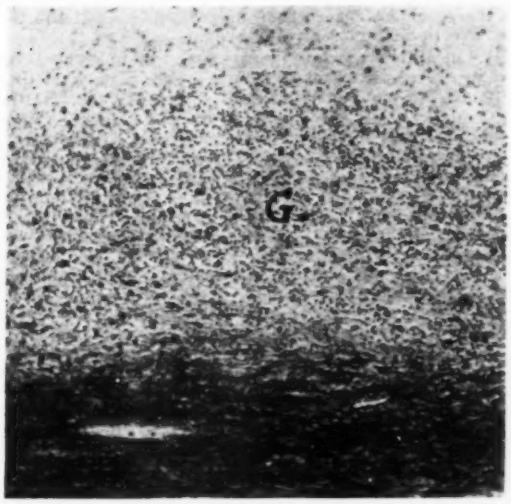


FIGURE X.

The granular layer G is slightly less dense than normal, but not unevenly, and the white centre is fairly normal (stained black); note that no Purkinje cells are to be seen. (Weigert-Pal preparation, × 150.)

90%:2·3%:7·8% in lieu of the normal 87%:2%:11%, if the whole brain is taken as 100%. The pia-arachnoid, while generally as usual for the man's age, yet over and behind the culmen this was greatly thickened; when the vermis was bisected, the clivus was seen to be atrophied and cystic more than elsewhere. This was proved microscopically. Transverse sections of the brain stem and pons compared favourably with norms; they were also larger than similar sections from the brother.

As only an oblique section of the medulla was preserved, one could only note that the olivary eminences were diminished. A hand lens revealed minute cysts, which were probably areas of atrophy, often around vessels. On the whole, this patient's intracranial contents were less affected than his brother's; less arteriosclerosis was in evidence.

FIGURE XI.

This cross section, the lowest level of the pons, next to the medulla, reveals fairly good staining of the inferior cerebellar peduncles R, R. (Weigert-Pal stain, × 4.)

Procedure.

Tissues were removed from the frontal, occipital and rolandic areas, from the hippocampus, the central nuclei, the brain stem, the pes and the pons (upper, middle and lower), and an oblique section was made through the medulla, the last few millimetres of the olives being included. From the cerebellum, sections were obtained

FIGURE XII.

Cross section of the upper third of the pons. The superior cerebellar peduncle S.C. is quite healthy, the transverse pontine fibres T.F. are equally so, as are the pyramids Py, and microscopically the pontine nuclei. A similar section of the cerebral peduncles showed good red nuclei and other structures. (Weigert-Pal stain, × 4.)

from the anterior crescentic lobule, the posterior superior and inferior lobules, the posterior crescentic lobule, the tonsil, bivalve and gracilis, as well as the uvula, central lobule, clivus, dentate nucleus and folia near by, and the inferior and middle peduncles separately.

The following stains were used: hæmatoxylin and eosin, Mallory's phosphotungstic hæmatoxylin, Mallory's triple

stain, picro-indigocarmine, Sudan III, Weigert-pal, iron hæmatoxylin van Gieson, Cajal's silver method, the Rogers-Foot method, and Bielschowski's, Nissl's and von Braunmühl's stains for argentophile plaques.

Neuropathological Lesions Found.

The slight thickening and remarkable absence of atheroma of the brain vessels was born out microscopically by the sections; a little pial thickening and subpial felting and some moderate thickening of the walls of the smaller cortical vessels were found. In places argentophile plaques were noted and glial cells and fibres were plentiful in the subcortical white matter—a common finding in the aged. Most of the neurones were well preserved, and both myelin and silver stains showed up myelinated fibres and axis cylinders not only associated with cerebral neurones, but in places or nides wherein perhaps hæmatoxylin and eosin might have indicated some neuronal decay. Some cortical neurones showed twisted principal dendrites.

Much of the forgoing applies to the central nuclei. In the brain stem, examination of the various nides there revealed good neurones such as those in the red nuclei and *substantia nigra*. The superior cerebellar peduncles were excellently preserved, as were the middle peduncles and pontine neurones; any cerebellar atrophy comprising the superior peduncles and the olivo-pontine-cerebellar type of Thomas was thus excluded.

Examination of the oblique sections from the medulla revealed good pyramidal tracts, as well as normal arcuate nuclei and tracts and fillets. Microscopically the minute cysts seemed to represent some perivascular atrophy or distension of certain areas around some blood vessels there, and a similar condition was also found in a portion of the clivus and on a smaller scale in the dentate nucleus. The condition was always quite local. The inferior cerebellar peduncles seemed fairly normal. On some aspects amyloid bodies were noted, as they were peripherally in the cerebral and cerebellar cortex and round the brain stem, and as these were followed some distance up the restiform body, it was thought that they might have indicated the course of the olivo-cerebellar fibres, many of which had atrophied. No granulations were found in the ependymal lining. However, glial fibres were abundant and common, forming a mesh round many of the larger vessels, and this was equally true for the white matter in the cerebellum. The optic chiasma and nerves appeared normal.

In Weigert-stained sections the olives were obviously pale, from the deficiency of myelinated fibres proceeding therefrom. With the hand lens the hilum was markedly devoid of issuing fibres—a fact well demonstrated by the paucity of fibres crossing the accessory olives to decussate. While the restiform bodies were slightly smaller and paler than normal, as they had been cut obliquely it was not ascertained whether this was due to the olivary cerebellar fibres already discussed. Many amyloid bodies were observed along these tracts as well as in the cerebral and cerebellar cortex. No healthy olive neurones were observed, but some pycnotic degenerate remains were noted, smaller than the hypertrophied glial cells which were a feature of this organ. Now Holmes points out that hypertrophied glia in nides severely deficient in neurones connotes that neurones were once there and had gone. A fair number of myelinated fibres seemed to exist in the olivary capsule, and the other tecto-olivary fibres seemed healthy.

The main features in the cerebellum are now discussed. The first feature was the almost universal disappearance of the ordinary healthy Purkinje neurones. In one paraffin section nine quite decent ones with antlers and Nissl bodies were noted, and a few elsewhere—and this in hundreds of paraffin and frozen sections. In their place were seen degenerate forms wherein the thin nucleus made up perhaps two-thirds of the structure and there was no sign of antler or axone. In common, too, with other cerebellar abnormalities, the position and orientation of the degenerated squibs differed from the more regular arrangements obtaining in healthy states. The squibs appeared dislocated, out of position, so that one readily mistook them for the various Golgi type II neurones which indeed were more conspicious and apparently more numerous, and outvied the Purkinje remnants in healthy prominence. While an excellent authority, Mullendorff, who quotes Cajal, maintains that these dislocated positions are within normal limits, actually in practice it is uncommon to see this phenomenon when the sections are cut perpendicular to the surface in well-fixed tissue.

In many varying pathological states, and especially at the apices of the folia, one may see a line of separation between the granular layer and the molecular layer. This is present in this cerebellum; the hypertrophied Bergmann glial cells are larger than the Purkinje squibs and send vertical fibres up to the pia intermingling with the down-growing fibres from the pial-glial junction. The Bergmann cells form quite a palisade between the granular and molecular layers. Gordon Holmes correctly points out that the Bergmann system (cells and glia) is not so prominent in heredo-cerebellar atrophy as in other cases. A good example of this is presented in the large Bergmann glial cells with broad bands staining like scarlet ribbons with Mallory's triple stain, in a case in which cerebellar nutrition was interfered with by the apposition of a cystic spongioblastoma.

The basket cell system—neurones, horizontal fibres and the baskets themselves—is so prominent (imbibition?) that the eosin stains render it easily seen. Von Braunmühl's silver stain shows this up in a remarkable manner. Ordinarily, in the sectioned basket thus impregnated is seen a yellow Purkinje body, with black side basket fibres surrounding it. In severe Purkinje cell atrophies, in the present case, the Purkinje neurones having degenerated to almost nothing, the baskets tend to collapse on them, and if the individual fibres are thickened as well, then examination of sections reveals almost solid bundles of basket fibres—empty baskets. Sometimes a pycnotic remnant may be seen in the basket.

Silver stains not only reveal the horizontal fibres of the basket cells and bodies, but also the various climbing fibres and extra glia when present, as in this case.

The granular layer may be slightly thinner than normal, but there are no spaces bare of granules. Still the Golgi type II cells are more readily seen.

The medullated white centre is well provided with myelin fibres, many of the thin variety, and Mallory's stains reveal many glial cells and perivascular gliosis.

The dentate nucleus neurones do not show up well in paraffin sections; yet in frozen Weigert-Pal sections processes in abundance are seen. Many small cysts apparently associated with the vessels of the nucleus sometimes interrupt the nerve cells. Whether alteration had taken place in the healthiness of the myelin capsule was not determined. However, the emerging fibres seemed thicker than usual.

Apart from the local pial thickening over the top of the vermis and the presumably arteriosclerotic degeneration of a few folia beneath this in the clivus, which with the anterior part of the *tuber valvulæ* shows some *état criblé* and even cyst-like cavities, one may briefly describe the chief lesions as a wholesale generalized degeneration of all the Purkinje cells of a centrifugal type, with greater prominence of the basket system and certain glia as well as a grave destruction of the olive neurones with severe compensating gliosis therein. These findings are common in heredo-familial cerebellar states.

It was a pity sections could not have been obtained of the cord and medulla to rule out the Marie and Friedreich types of atrophy. Wertham gives good diagrams disclosing the centripetal type of lesion occurring in more general pathological states; the plumes are prominent, while the basket system degenerates and fades into insignificance.

Finally, silver stains revealed plenty of axis cylinders in the granule layer, and any thinning of the granules made no more prominent those bare areas found there which were once thought to be structures of importance; but now these so-called acidophile structures of Denissenko are considered synapses (Mullendorff).

Comment.

This clinical record is published in the hope that it may help in the discovery of similar examples of this condition, both from this family and from others. All authors describing this disease and writing upon the subject are agreed that many more records are required before classification of this type of cerebellar disorder can be undertaken satisfactorily. After reading most of the literature of this subject, we agree with writers who testify to its confused state at the present time.

Examples of heredo-familial disease resembling that described here have been published in English by Holmes,[3] by Weber and Greenfield,[4] and by Akelaitis.[5] In all of these families gross cerebellar disorder was present, which affected chiefly the lower limbs, and the outstanding pathological feature was a generalized atrophy of Purkinje neurones of the cerebellar cortex. Degeneration of cells of the olives was also present, but this was secondary to that of the cerebellum. Variations exist in the clinical pictures of these families, in that sexual underdevelopment was a feature of the patients described by Gordon Holmes; acute mental disorder with convolutional atrophy and loss of third and fifth cortical cell layers in frontal and parietal regions was present in the two patients described by Akelaitis; a more chronic mental deterioration accompanied the closing stages of the illness of Weber and Greenfield's patient; while in the family we have described neither sexual hypoplasia nor mental disorder existed.

These minor variations do not prevent these families from being classed under the same heading, as in their broad outlines the clinical records are similar.

According to the classification of Gordon Holmes (1907), these families belong to Class I "primary parenchymatous degeneration of the cerebellum". Marie, Foix and Alajouanine classified the disease under the headings of "congenital", "acquired" and "heredofamilial"; in such a grouping the last category would include the families reported. However their conditions are classified, these patients manifest a progressive disorder of the cerebellum which occurs in families throughout several generations, with Purkinje cell atrophy of the cerebellar cortex as the most prominent pathological feature.

Purkinje cells are vulnerable structures. They do not reach maturity until the second year of life, and like all nervous tissues which develop late, they are prone to be the first to be affected by disease or degeneration. Commencing degeneration of these cells is seen normally soon after the age of forty years, and this degeneration progresses with the years. It is said to be most prominent in the anterior part of the cerebellum. Observations have been made by one of us (O.L.) of Purkinje cells in the cerebella of patients who had died of tuberculosis, cerebellar tumour, encephalitis (acute and chronic), *dementia paralytica* and septicæmia, the last-mentioned having been treated with a sulphonamide. In the cerebellum of the patient who had died from septicæmia, all gradations from normal to pycnotic nuclei of cells were seen. Numerous miliary abscesses were present, and the cells close to these were badly affected. In all the above conditions antlers of Purkinje cells were frequently missing, and Purkinje cells which had lost normal globular shape or showed scarcely any cell body and whose nuclei stained poorly were commonly seen. Similarly, many examples of cerebellar disease associated with widespread degeneration of Purkinje neurones have been reported with such varying ætiological factors as syphilis, chronic alcoholism, lead, infections such as typhoid fever[7] and conditions described as enteritis.[8][9] In such enteric disorders it is possible that the nervous symptoms and the bowel symptoms may both have been the result of a virus infection, rather than that the cerebellum had been affected by toxins from the bowel. In this connexion it is interesting to note that the virus of louping-ill, a disease of sheep, is capable of producing a degeneration of the Purkinje cell layer. If abiotrophies of the cerebellum are thought to be primary degenerations, we consider the examples mentioned above as constituting secondary parenchymatous degeneration because of the damaging effect known to follow such neurotrophic toxins.

Clinical reports of a condition characterized by extensive Purkinje cell atrophy have been published under such varying titles as "Parenchymatous Cortical Cerebellar Atrophy" (Parker and Kernohan[10]), "Lamellar or Sclerotic Atrophy of the Cerebellum" (Hassin[11]), "*De l'atrophie cérébelleuse tardive à prédominance corticale*" (Marie *et alii*[6]). These reports have been collected and reviewed by Parker and Kernohan,[10] and by S. A. K. Wilson.[12] Kinnier Wilson has described them under the heading of "intracerebellar and cerebellifugal types of cerebellar atrophy". The absence of heredo-familial characteristics appears to indicate that these patients did not suffer from a primary degenerative cerebellar disorder. These authors seem to have considered them to belong to a secondary group, as ætiological antecedents have been described. Admittedly these antecedents, as Wilson remarks, "can only be described as vague". It is possible that some of the cases described may be gathered later into a primary familial group of cerebellar degeneration by further study of the families concerned.

Finally, we agree with Weber and Greenfield that the primary familial group, in which their case and ours fall, differs entirely from those examples of cerebellar disease described by Hassin and Harris as "olivo-ponto-cerebellar atrophy".[13] In this type of atrophy not only is the onset of the disorder in early life and its duration much shorter, but the pathological picture is entirely different, in that the outstanding changes are to be found in the cells of the medullary olives, the arcuate nuclei and the pons. Purkinje cells are only slightly affected through a trans-synaptic or retrograde degeneration of cerebello-petal fibres.

Summary.

1. The clinical record and the pathological and histological findings in the cerebellum of a patient who died of familial cerebellar disease are given. This is the second case to be reported in this family.

2. The outstanding feature was widespread atrophy of the Purkinje cells of the cerebellar cortex.

3. Conditions associated with this form of degeneration are discussed, and are divided into primary and secondary groups. The former is thought to be an example of abiotrophy and the latter the result of a toxi-infective process.

4. The families described by Gordon Holmes, by Weber and Greenfield and by Akelaitis and ourselves are considered to be instances of the same pathological process.

Acknowledgements.

Acknowledgements are due to Professor Keith Inglis, by whose efforts a continuance of the work done in the old Mental Hospitals Department laboratory has been made possible; to Professor A. N. Burkitt for anatomical facilities; to Mr. Woodward Smith for his photographs and for many suggestions thereon; and to Miss McCulloch, B.Sc., for the preparation of some of the sections.

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BLOOD GROUPS, M, N TYPES AND THE Rh FACTOR IN INDONESIANS.

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THE blood group frequencies of Indonesians are well known, as the result of extensive investigations by Buining, Bijlmer, Lehmann, Grove and others. The results of these surveys have been tabulated by Boyd (1939). The B factor was found in all samples, and while there was much variation in the percentage, in most surveys 20% to 40% of the samples belonged to group B. No test was made for the subgroups of A, and no reference has been seen to date showing that they have been determined.

Birdsell and Boyd (1941) make the following comment in their paper dealing with blood groups in Australian aborigines:

> It is unfortunate that we have no reliable data for M and N (where differences between Australia and North America are most striking) for the regions through which the early populations must have passed. There is a gap in our knowledge extending from Cape York Peninsula to Hong Kong in the former case, and from Montana to Japan in the latter. It would be most desirable to have M, N data for Melanesia, Indonesia, and for the marginal populations of the eastern Asiatic mainland.

Boyd (1939) has listed three M, N surveys on Indonesians reported by Postmus (1934), and subjected the results to statistical analysis. The figures he obtained for D/σ—6·16, 7·67, 8·67—in each survey suggested that the results obtained by Postmus were unreliable. The three surveys were made on blood samples from Batavians, Javanese and Sundanese when Postmus was stationed in Batavia.

In two surveys carried out in Melbourne (Wilson, Graydon, Simmons and Bryce, 1944, and Simmons, Graydon and Hamilton, 1944) are reported the results obtained with the blood of Australian aborigines. The blood groups and M, N figures confirm the earlier work of others, and in addition it was found that all the blood samples of group A examined were of subgroup A_1 and that all blood samples tested were Rh-positive. After these surveys it was decided that as opportunity permitted we should carry out blood surveys on Indonesians, Melanesians and Polynesians and thus obtain new information concerning the subgroups of A, the M, N types and the Rh factor for native populations adjacent to Australia.

This report deals with the results obtained with Indonesian blood. New data which we have obtained for Papuans, Fijians and Maoris will be the subject of later reports.

Materials and Methods.

The blood samples were collected by us in or near Melbourne from Indonesian soldiers, sailors and civilians. The difficulty of collecting blood samples from only pure-blooded Indonesians is well known, owing to admixture with other races which has taken place for several hundred years. Each person from whom a blood sample was taken was scrutinized by one of us (C.O.), who had spent a number of years as a medical officer in the Dutch East Indies. Each was questioned as to his island of origin and also as to the island on which his mother and father were born, and as a result a few persons were excluded from the series.

Owing to the absence of family relationships amongst these adult natives, and to the fact that they originated on a number of islands and from many different districts, it is thought that our sampling probably represents a cross-section of the Indonesian people as they are today.

The blood samples were nearly all examined within 24 hours of collection and all tests for groups, types and the Rh factor were carried out by the slide technique as described by Simmons *et alii* (1941 and 1943).

Results.

The natives whose blood we examined came from Java, Timor, Celebes (Menado), Ambon and sixteen other islands. The details of islands, districts and the blood group frequencies are shown in Table I.

The outstanding feature of the grouping results is the absence of the subgroup A_2, despite the presence of the A_1 factor in eighty of the samples. Otherwise the blood group picture is similar to that obtained from the many previous surveys on Indonesians.

In Table IIA are shown the MN results for this survey, and for comparison the Indonesian results of Postmus are given in Table IIB. The m and n frequencies found by us do not differ greatly from those reported by Postmus, but we were unable to confirm the low frequency of type MN found by him. There is more than a suggestion of a gradient in the M, N figures, the frequency of "n" being low in Java and high in Timor.

Of 296 blood samples tested for the Rh factor, 294 were Rh-positive. Three of the samples failed to react with human anti-Rh serum "Mrs. C.", which, when used as a preliminary test serum, reacted with blood from 82·4% of a total of 3,641 white Australians (Simmons *et alii*, 1943). One of these three samples reacted with another human anti-Rh serum "Mrs. P.". The remaining two samples were not agglutinated by any of seven other human anti-Rh sera and an immune guinea-pig serum, and so can reasonably be considered to be genuinely Rh-negative.

Known control blood samples were used as a check during the entire investigation, and as a routine measure any blood sample which was found to be AB, MN and Rh-positive was then retested with normal human serum of group AB, to exclude the possibility of panagglutination. No sample showing panagglutination was encountered.

Discussion.

The lack of subgroup A_2 in Australian aborigines, in American Indians, in Hawaiians and probably amongst pure Chinese and now in the Indonesians, indicates that the determination of the subgroups of A amongst native races will become a point of greater importance for studying racial origins. The same also applies to any new data presented for M, N types and for the Rh factor and its subtypes.

The Australian aborigines all have Rh-positive blood, and this also applies to American Indians and Chinese, and now to the Indonesians, with only occasional exceptions which are probably due to undetected white admixture. In our series the two Indonesians whose blood was Rh-negative were unrelated; both came from different districts in Celebes (Menado). Both men were critically examined by Dr. H. van Duyl, who reported that he could detect no trace of racial admixture. Inquiries made through the Netherlands East Indies Information Service concerning the early settlement of Menado brought the following reply:

[1] Captain, Netherlands East Indies Army.

TABLE I.

Blood Group Frequencies in Indonesians.[1]

Island.	Number Tested.	Number of Districts.	Number and Percentage of Groups.				Frequency of Genes.			D/σ_D.
			O.	A_1.	B.	A_1B.	*p*.	*q*.	*r*.	
Java	107	32	45 (42·1)	27 (25·2)	27 (25·2)	8 (7·5)	0·180	0·180	0·649	0·6
Sixteen other islands	61	29	31 (50·8)	13 (21·3)	14 (23·0)	3 (4·9)	0·141	0·151	0·713	0·32
Celebes (Menado)	47	24	33 (70·2)	6 (12·8)	7 (14·9)	1 (2·1)	0·078	0·089	0·838	0·53
Ambon	43	19	22 (51·2)	16 (37·2)	5 (11·6)	0 (0)	0·208	0·060	0·716	1·14
Timor	38	3	22 (57·9)	5 (13·2)	10 (26·3)	1 (2·6)	0·082	0·157	0·761	—
Total	296	107[2]	153 (51·7%)	67 (22·6%)	63 (21·3%)	13 (4·4%)	0·146	0·138	0·719	0·44

[1] The gene frequencies were derived from the following equations:

$$p = 1 - \sqrt{\frac{O + B}{100}}$$

$$q = 1 - \sqrt{\frac{O + A}{100}}$$

$$r = \sqrt{\frac{O}{100}}$$

$D = 1 - (p + q + r)$. $\sigma_D = \sqrt{\frac{pq}{2V(1-p)(1-q)}}$, where V = number of individuals in each sample.

[2] Incomplete data.

TABLE IIA.

M, N Frequencies in Indonesians (Present Survey).

Island.	Number Tested.	Number and Percentage of Types.			Frequency of Genes.		D/σ_D.
		M.	MN.	N.	*m*.	*n*.	
Java	107	51 (47·7)	44 (41·1)	12 (11·2)	0·682	0·318	0·52
Sixteen other islands ..	61	17 (27·9)	26 (42·6)	18 (29·5)	0·492	0·508	1·1
Celebes (Menado)	47	8 (17·0)	23 (48·9)	16 (34·0)	0·415	0·585	0·06
Ambon	43	8 (18·6)	25 (58·1)	10 (23·3)	0·477	0·523	1·1
Timor	38	6 (15·8)	17 (44·7)	15 (39·5)	0·382	0·618	0·33
Total	296	90 (30·4%)	135 (45·6%)	71 (24·0%)	0·532	0·468	1·42

The gene frequencies were calculated from the relations $m = \frac{2M + MN}{2V}$, $n = \frac{2N + MN}{2V}$, $D = 1 - \left(\sqrt{\frac{M}{V}} + \sqrt{\frac{N}{V}}\right)$, $\sigma_D = \frac{1}{2\sqrt{V}}$, where V = number tested.

TABLE IIB.

M, N Frequencies in Indonesians Found by Postmus (1934).

People.	Number Tested.	Percentage of Types.			Frequency of Genes.		D/σ_D.
		M.	MN.	N.	*m*.	*n*.	
Batavians	132	38·0	19·5	42·5	0·48	0·52	6·16
Javanese	194	36·0	14·0	50·0	0·43	0·57	8·55
Sudanese	198	40·5	14·5	45·0	0·48	0·52	8·65

I can advise you that the first evidence of Dutch being in Menado dates from the year 1657, when Simon Cos built a fort near that place. Before that time the Spaniards had already been in Northern Celebes, but were driven out by the Dutch.

Owing to the early date of settlement, particularly in Menado, it is probably not surprising that the blood of two natives in this series proved to be Rh-negative.

Our M, N results are similar to those previously reported for Japanese and Chinese. They differ considerably from the results of Postmus in the frequency of type MN. Statistical analysis ($D/\sigma_D = 1·4$) suggests that our results are reliable.

When Postmus made his surveys only a few other M, N figures had been reported, and at that time there was no established statistical test by which he could check his results. He had much difficulty in obtaining preliminary testing fluids; his first lot of absorbed fluids from Warsaw were found to be useless on arrival. Later he states that he obtained, also from Warsaw, unabsorbed sera which he absorbed satisfactorily and used as the basis for the preparation of his own testing fluids. It is known that weak testing fluids may fail to react with some samples of MN cells while giving firm reactions with cells of the homozygous types M and N. We suggest this as a likely explanation of the extraordinarily low MN percentage

found by Postmus in Indonesians. He also obtained similar results when testing the blood of Hollanders at the same time, and to date no further M, N investigations have been reported on this race to confirm or disprove the results obtained by him. When this is done, it is to be anticipated that the low MN figure will be corrected without any appreciable alteration in the relative frequencies of the genes as found by him.

Summary.

1. Blood samples from 296 Indonesians who originated on various islands in the Dutch East Indies have been grouped, subgrouped, M, N typed and tested for the Rh factor.

2. The percentages were: group O, 51·7; group A, 22·6; group B, 21·3; and group AB, 4·4. All of group A and AB (80) were of subgroup A_1 and A_1B.

3. The percentages were: type M, 30·4; type MN, 45·6; and type N, 24·0. These figures differ greatly from those recorded by Postmus in 1934.

4. Of the 296, 294 samples were Rh-positive (99·3%); this indicates that probably all pure-blooded Indonesians have Rh-positive blood. The two persons with Rh-negative blood both came from Celebes (Menado), and as this area was one of the first to be settled by the Spanish and then the Dutch, it is possible that their blood was Rh-negative owing to some untraceable white admixture.

Acknowledgements.

We are grateful to Dr. F. G. Morgan, Director of the Commonwealth Serum Laboratories, and to the Dutch authorities for permission to carry out this blood survey, to Dr. H. van Duyl and Mr. P. L. Jansse, who cooperated with us in the collection of blood samples, and to Mr. D. J. Simper, B.Sc., and Mr. E. F. Woods, B.Sc., who worked with us during part of this investigation.

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FROM WHAT DID SUDDS DIE?

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THE Lieutenant-General regrets it has become his duty to expose to the indignation of the troops, two soldiers, who have committed an offence under circumstances which leave little doubt that their object was to obtain their discharge from the service.

Private Joseph Sudds and Patrick Thompson, of the 57th regiment, have been convicted of a robbery in the town of Sydney, committed in open day, and without even an attempt to conceal the fact, and have been sentenced to be transported to a penal settlement for a period of seven years.

The Lieutenant-General, in virtue of the power with which he is vested as Governor-in-Chief, has thought fit to commute the sentence, and to direct that privates Joseph Sudds and Patrick Thompson shall be worked in chains, on the public roads, for the period of their sentence, after which they will rejoin their corps.

The above words form the opening paragraphs of a general order issued from army headquarters in Sydney on November 22, 1826. They form the introduction to one of the most notorious incidents of early Australian history. On several occasions previously soldiers had committed offences with the idea of being transferred from military duty to what they considered to be the easier life of a convict. The Governor, Lieutenant-General Ralph Darling, was determined to stamp out the practice. Accordingly he devised an unusual form of punishment. On November 22, 1826, the two men were taken from the gaol where they had been since their trial on November 8 and were marched to the barracks. Both buildings were in George Street. There, in front of the assembled regiment, they were stripped of their uniforms. When they were naked, a wheelbarrow was wheeled into the square in which were certain irons and blacksmith's apparatus. There were also taken from this barrow yellow jackets, yellow trousers and yellow caps. This yellow clothing was then put on the two soldiers. As soon as the clothing was on the men were laid down and bound and their heads were placed on small blocks. The irons were taken from the wheelbarrow and were riveted on by the blacksmith. It was then seen by those present that the irons were unusual. They consisted of an iron collar round the neck with two projecting spikes from six to nine inches in length. From each side of the collar there were attached chains which went down to the basils on the ankles. It was subsequently stated by the Government's opponents that the irons were so arranged that the men could neither lie at full length nor rest their heads. It was also said that the irons were excessively heavy. The felon's clothing and the irons having been put on, the men were marched to the barrack gate preceded by two drummers playing the "Rogue's March". They were then taken through the crowded street back to the gaol. It was a very hot day. The degradation took place on a Wednesday. On the following Monday Sudds died. There was at once a tremendous public outcry, the echoes of which did not die down till ten years had passed.

It was alleged, of course, that Sudds had died as the direct result of an inhuman punishment. The putting on of the chains was undoubtedly the immediate cause of death. The records show, however, that Sudds was already a sick man when the punishment was inflicted. The Government did not know of this, and if there is any blame to be attached to anyone, it must be attached to the surgeon, Dr. McIntyre, who was well acquainted with Sudds's history, and who completely under-estimated the seriousness of the illness. The purpose of this paper is to present the available evidence concerning Sudds's illness. The main reason of this is that the medical history is interesting. Another reason, however, is to allow justice to be done to Governor Darling. Most of those who have commented on this incident (of whom Marcus Clarke is the most famous) have assumed that General Darling was very much at fault. Wentworth actually accused him of murder. It is doubtful whether this view is correct. There is no doubt that the Governor did not know how seriously ill Sudds was. In regard to the punishment itself, it may be stated at once that it was never proved that the men could not lie down properly because of the chains, and it was never proved that they were excessively heavy. It has to be remembered also that chaining was as common in those days as imprisonment is in ours. It is even on record that women were put in iron collars with projecting spikes to prevent their going through windows. It will be generally admitted that some unusual and striking form of punishment was necessary in the case of Sudds and Thompson.

The following evidence in regard to Sudds's health was given before the Executive Council.

John Toole, first turnkey, stated that "Sudds was taken ill a few days before he was tried; he was swelled very much from his head to his feet".

John Thompson, medical attendant at the gaol, made the following statement:

> In the early part of November, Sudds was ill and was an outpatient; he was bled and took opening medicine. Some days after, about nine o'clock in the evening, he was brought into the sick ward, and was very ill; he was much swollen; he complained of his head and bowels; I made him foment his belly on the following morning with hot water; it removed the pain and swelling from his belly, but the swelling in his legs and thighs still continued. When the surgeon came in the forenoon, he ordered him to be discharged from the hospital; his head and bowels were then better, but the swelling of his legs and thighs still continued. He mentioned that he supposed it to be dropsy, the same complaint he had formerly had an attack of in the West Indies. He remained out of hospital a few days, when he again returned in a much worse state than he formerly was; I made him have recourse to the same fomentations and opening medicines. I asked him when he came into hospital, what is the matter with you? He said, my belly is like a drum. He remained in hospital with the gaol-irons on until the morning of the 22nd; the irons were then struck off, and he was taken to the barracks. On his return he was again put to the "fines room"; the next day (Thursday the 23rd), between twelve and two, he was brought to the hospital and placed in bed. Mr. McIntyre saw him immediately, and ordered his irons to be taken off; Mr. Toole, to whom this order was given, replied that the irons had been put on by direction of the Government, and he could not remove them. Mr. McIntyre observed, the irons are too heavy to be worn here, and they must be taken off whilst he is in hospital, at any rate. About four o'clock that afternoon, some time after Mr. McIntyre went away, the irons were taken off. Sudds continued to get worse, and was removed to the general hospital on Sunday afternoon. He had complained from the first time he was taken ill, in the beginning of November, of a pain in the throat, and of a difficulty in breathing; which difficulty increased to such a degree that he could scarcely breathe, and was totally unable to take nourishment after Friday, when he took in my presence a little arrow root and tea. On Saturday he was unable to swallow anything but a little water, which I put into his mouth with a spoon; and this inability to take food continued until his removal.
>
> From the time of his first coming into the gaol hospital early in November, he had no appetite; he scarcely ate anything, and appeared to have an incessant desire to drink.

Mr. McIntyre, the assistant surgeon, made the following statement:

> I saw Sudds on the 11th November when he had swelling in his legs, and was placed in the prison hospital. He took medicine until the 16th, and seemed better; I mentioned to him that I did not think there was anything the matter with him. I discharged him from the hospital, and did not see him until the 24th November, when he again applied to me; from the symptoms I observed, I imagined that he might be affected with dropsy, and interrogated him accordingly. I had not heard of his being ill before that day; he was taken into hospital on the 24th, and was treated by me; I ordered his irons to be taken off; I saw him on the following day, and found him very low; I again examined his body and did not discover any material symptoms, further than the continued swelling in his legs; he had medicine administered to him on that occasion, and his breathing was quicker than usual, and his spirits were oppressed. On Sunday morning I again visited him, and ordered him to be immediately removed to the general hospital; he was then in a delirious state. I saw him occasionally between this time and the period of his death. On opening the body, I first examined the abdomen, which appeared quite healthy; I then examined the liver, which seemed to me larger than in general; it was healthy in all other respects.
>
> I then examined the head, and found the brain quite healthy; I then examined the throat, and discovered mucus of a slimy frothy description; the windpipe was rather inclined to a reddish colour.

Mr. McIntyre stated that the following was the exact conversation he had with Sudds on the day of his second admission into the hospital (November 24):

> I said to Sudds, you have brought yourself into pretty disgrace, you will be a fine figure with those irons at work; he replied, "I will never work in irons". I said, it would be better for you to be out of the world; he replied, he wished to God he was.

It turned out that when McIntyre had previously reported to the Governor on the result of the post-mortem examination, his account of the findings in the chest was different from his later account. He had then said that there was an inflammation or inflammatory appearance extending from the chest upwards to the throat, where it was more extensive, and which he would term "bronchitis".

Mr. McIntyre was asked to produce the hospital books, but he said that none were kept. He was asked to produce the record of the post-mortem examination, and said none had been kept.

Dr. Bowman, the principal surgeon, made the following statement:

> About a fortnight or three weeks previously to Sudds' death, I visited the gaol with Mr. McIntyre, the assistant surgeon, who had the charge of the gaol, and found that Sudds had been treated for dropsy, but was that day discharged from the sick list, his disease having subsided. I never saw him in gaol but upon the above occasion. He was sent to the General Hospital on Sunday evening, I think the 26th of November, 1826, about five o'clock, upon Mr. McIntyre's application to the sheriff to have him removed in consequence of his severe illness. I saw him when he was received into the General Hospital, and again on the following morning. He was in a state of delirium, and unconscious of anything that was said to him. I understood from Mr. McIntyre that Sudds had refused every sort of sustenance for several days, and in the state of exhaustion so occasioned delirium was produced. In fact, he had determined to die.

Mr. Mitchell, surgeon of the general hospital, was present at the post-mortem examination and stated:

> The body in general appeared healthy, and no particular organ was so materially affected as might be justly called the proximate cause of death; the liver seemed to indicate that he had lived freely, but it was more functional derangement than organic disease.

Thompson, who was punished with Sudds, stated:

> Sudds was turned out of the hospital on the morning of the punishment, and taken to the barracks about an hour afterwards. He was taken from the hospital to the Sessions, on the 6th of November; he appeared to be very ill, insomuch that the man who was handcuffed to him was obliged to sit down on the grass in the court-yard, in order to enable him to lie down. I, for the most part, got Sudds' rations of bread; he could not eat it himself. On our return to the same ward in the gaol (immediately after the punishment), Sudds sat down with his back to the wall, saying that he was very ill, and wished to go to the hospital again; but he did not go to the hospital till the next morning.

The following is a summary of Sudds's illness: he had had a previous attack in the West Indies; this was in about 1800. On November 4, 1826, he complained of illness; on November 8 he was tried; on November 11 he was placed in hospital with dropsical symptoms; on November 16 McIntyre discharged him from hospital and ordered the irons to be placed on him; on November 19 he was readmitted to hospital, with the irons still on; on November 22, the degradation took place; on November 24 he was again admitted to hospital and the irons were removed; and on November 27 he died.

Discussion.

It is obvious that not enough information is available to enable an exact diagnosis to be made. Sudds may have had cardiac failure, in which case, as scarring of the valves is not mentioned, it was probably arteriosclerotic. He may have had Bright's disease. He may have had cirrhosis of the liver. If, however, the dropsy is taken as a genuine recurrence of the attack he had whilst the regiment was in the West Indies, none of these diagnoses

is tenable, as it is impossible to imagine that he would have been free from dropsy for over twenty years, or indeed that he could have lived for so long. If it is assumed that the recurrence was a true recurrence, is there any other possibility? It has been suggested to me by Dr. T. A. F. Heale, of Melbourne, that Sudds may have been a drunkard and may have been suffering from vitamin *B* deficiency. A further search of the records made by the Mitchell Librarian has revealed that Sudds was indeed a drunkard. Lieutenant-Colonel Thomas Shadforth, of the Fifty-Seventh Regiment, stated:

> Sudds' conduct as a soldier was considered bad. He was a drunkard. When the irons were put on Sudds, he appeared to be in a sickly state, but I attributed it to his having been a hard drinker and having been deprived of the stimulus to which he was used.

Although there is no mention of the neuritic symptoms which often accompany vitamin *B* deficiency, its possibility has to be admitted, particularly as the diagnosis would fit in with a previous attack such as Sudds may have had. The exact diagnosis of Sudds's case must, however, remain a matter for conjecture.

Of more interest, however, than "from what did Sudds die?" is "what should McIntyre have known?"; in other words, what was the state of contemporary knowledge? McIntyre's mistake was a serious one for Governor Darling. There was a large body of free men and emancipists in the colony who were bitterly opposed to Darling's autocratic rule. They were led by Wentworth, who fanatically regarded the struggle as one for constitutional freedom. The incident which has been described was made the most of by this faction. Indeed, they made so much of it and of other instances of autocratic rule that in the end Darling was obliged to resign. The question arises, therefore, "should not McIntyre have had sufficient clinical knowledge to realize that dropsy was a most serious symptom?" The answer is that of course he should. Dropsy is one of the classical symptoms in medicine, and ever since there have been patients and doctors interested in them, so long must dropsy have been under discussion. As a matter of fact, as will be shown shortly, the period of this incident was one of those interesting periods of medical history when the various diseases having in common a particular syndrome were being separately recognized. The state of knowledge with regard to dropsy in the latter half of the eighteenth century is shown by a study of "First Lines of the Practice of Physic", by William Cullen, M.D., published in 1786. His discussion "Of Watery Swellings or Dropsies" is masterly. He opens the discussion with the following statement:

> In persons in health, a serous or watery fluid seems to be constantly poured out, or exhaled in vapour, into every cavity and interstice of the human body capable of receiving it; and the same fluid without remaining long or being accumulated in these spaces, seems constantly to be soon again absorbed from thence by vessels adapted to the purpose. From this view of the animal œconomy, it will be obvious, that if the quantity poured out into any space, happens to be greater than the absorbence can at the same time take up, an unusual accumulation of serous fluid will be made in such parts; although the quantity poured out be not more than usual, yet if the absorption be any wise interrupted or diminished, from this cause also an unusual collection of fluids may be occasioned.
>
> Thus, in general, dropsy may be imputed to an increased effusion, or to a diminished absorption; and I therefore proceed to inquire into the several causes of these.

If the rest of the discussion was summarized in the modern fashion, it would read as follows:

A. A Preternatural Increase of the Ordinary Exhalation.

1. An interruption given to the free return of venous blood from the extreme vessels of the body.
   (i) A polypus in the right ventricle.
   (ii) Ossification of the valves.
   (iii) Obstructions in the vessels of the lungs preventing the entire evacuation of the right ventricle.
   (iv) Scirrhosity of the liver preventing blood flowing into it from the *vena portarum*.
   (v) Pressure of tumours directly on veins.

2. Increased exhalation due to laxity of the exhalant vessels.
   (i) Paralysis of the limbs.

(ii) General debility of any sort.

(iii) "Among other causes inducing a general debility of the system, and thereby dropsy, there is one to be mentioned as frequently occurring, and that is, intemperance in the use of intoxicating liquors; from whence it is that drunkards of all kinds, and especially dram drinkers, are so affected with this disease."

The last paragraph is so important that it has been quoted as in the original. It shows that the physicians of the period had remarkable powers of clinical observation. At this period, one hundred and fifty years before the discovery of vitamins, the circumstances that are known to accompany lack of vitamin *B* and vitamin *C* had already been noted. In the case of vitamin *C* deficiency, a method of prevention and cure had also been discovered.

3. Increased exhalation due to a preternatural abundance of serous or watery fluid in the blood vessels, especially when such abundance concurs with the causes above mentioned.

(i) An unusual quanity of water taken into the body.

(ii) An increase produced by the skin changing from a perspiring to an imbibing state.

(iii) An interruption or diminution of the urinary secretion as in the case of *ischuria renalis*.

This paragraph shows that the possibility that renal disease might cause dropsy was already being considered. As will be seen shortly, this conjecture was soon shown to be fact.

4. The spontaneous or artificial evacuation of large quantities of blood. "These evacuations by abstracting a large proportion of red globules and gluten, which are the principal means of retaining serum in the vessels, allow the serum to run off more readily by the exhalants."

The dropsy often attending chlorosis is probably due to a fault of digestion, whereby, although the watery parts of the food pass in, the materials for the production of blood and gluten are imperfectly absorbed.

This is an interesting assumption, in view of the fact that the cure for chlorosis was later found to be the giving of large quantities of iron.

B. Diminished Absorption.

Cullen in this connexion names no specific causes, but supposes that the same general causes which would produce increased laxity of the exhalants would at the same time produce diminished tone in the absorbents.

A study of this article shows that at the end of the eighteenth century physicians were, with the exception of Bright's disease, well aware of the main causes of dropsy. Obviously, furthermore, a good deal of study had been given to the physiological side of the problem.

In 1812 and 1813 there appeared the remarkable studies of Wells and of Blackall, both of whom noted the association of dropsy, albuminuria and contracted kidneys.

In 1827 appeared Bright's "Report of Medical Cases", which contained his masterly account of the disease now known by his name.

Enough has been written to show that a well-trained doctor in the year 1826 should have known that dropsy was a most serious symptom.

Conclusion.

From 1826 to 1836, when the last parliamentary inquiry into the affair was held, Sudds and Thompson could hardly have been out of Governor Darling's mind for a single day. Quite apart from the general onslaught on him, headed by Wentworth, he was involved in a further affair with an officer named Robison. Robison was present at the degradation of the two soldiers, and later tried on Sudds's irons. He severely criticized Governor Darling, and as a result was court-martialled and cashiered. His petitions for reinstatement occupied the authorities for years. He published in London a pamphlet, the cover of which is shown. Darling sued him for libel and he spent three months in the Marshalsea prison. He did not succeed in any of his petitions.

Thompson's chains were taken off after a short period and he was pardoned in 1829.

Acknowledgements.

I am indebted to Mr. Justice Ferguson and to the Mitchell Librarian for personal communications, and to Dr. R. Wishaw for the photograph of the pamphlet.

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"The Historical Records of Australia."

Reviews.

TREATMENT IN PSYCHIATRY.

IN "Physical Methods of Treatment in Psychiatry", Dr. W. Sargant and Dr. E. Slater have described the techniques which they found both practicable and effective in emergency hospitals for psychiatric casualties.[1] Rather ingenuously they state that "these methods have served us well as a selected battery for the treatment of the early and recoverable cases seen at the Maudsley and at Sutton". No doubt some sceptics will ask: "What is the real value of some of these dangerous and complicated therapies in 'early and recoverable' cases?" The authors have not attempted any statistical analysis of their results, but somewhat dogmatically record their impressions and lay down the indications for the methods selected in different types of case. Although this is not a general work on psychiatry, the introduction contains an interesting exposition of the authors' views on ætiology and a general survey of their method of approach to psychiatry. Here it is clearly seen that they were students of the late Professor Mapother, to whose memory the work is dedicated.

Insulin-coma therapy is described at length and is strongly recommended for schizophrenics whose illness has been rapid in onset and of only a few months' duration. In spite of their opinion, "the factors that favour a satisfactory response to insulin are those clinically associated with a higher expectation of spontaneous remission", the authors consider that there are enough tragedies from the neglect of insulin therapy to justify the administration of this treatment in selected cases. Convulsion therapy is often indicated as an adjunct and may be all that is required in the case of those who display a schizophrenic symptomatology combined with more than a moderate degree of inhibition or depression. In view of their enthusiasm for insulin-coma therapy, it should be noted that the authors' experience has been not with patients under certificate, but with "early" and selected patients treated on a voluntary basis. They believe, however, that they have arrested the downward course in a significant proportion of cases. It is this question of the duration of symptoms and indeed of the actual diagnosis (especially from toxic-exhaustion and depressive states) which renders the comparison of sets of statistics so full of fallacies.

Modified insulin therapy, the fattening process, is a development of the Weir-Mitchell method. The authors quote some of Weir Mitchell's observations on his experiences in the American Civil War and on the evolution of the *régime* which bears his name. It would have been appropriate to refer to the letter from Weir Mitchell to Osler quoted in Cushing's "Life" in which Mitchell reported the first and successful application of his method to an hysterical woman in 1874. The use of insulin as an appetizer is most effective in the patient whose previous personality has been adequate, but who has suffered exceptional stress with consequent loss of weight and secondary symptoms referred to the intellectual or emotional systems. While such cases always bear a good prognosis, insulin-fattening reduces the period of

[1] "An Introduction to Physical Methods of Treatment in Psychiatry", by William Sargant, M.A., M.B. (Cantab.), M.R.C.P., D.P.M., and Eliot Slater, M.A., M.D. (Cantab.), M.R.C.P., D.P.M.; 1944. Edinburgh: E. and S. Livingstone Limited. 8½" × 5½", pp. 181. Price (English): 8s. 6d. net.

invalidism. Convulsion therapy usually leads to a quicker disappearance of inhibition and depression, even in the catatonic syndrome, than is the case with other methods. Some schizophrenics appear to be worse after convulsion therapy, especially hebephrenics, who also react unfavourably to insulin coma. Emphasis is laid on the importance of keeping patients under observation after discharge from hospital in order that treatment may be repeated at the earliest indications of a relapse.

In a chapter on cerebral dysrhythmia the authors discuss the relationship between epilepsy and certain forms of temperamental instability, such as phases of agressiveness, impulsiveness, irritability or dulness, in which an abnormal electro-encephalogram has been demonstrated and improvement has followed the exhibition of anti-convulsant drugs. Such a relationship has long been recognized in the light of clinical experience including studies in heredity, but is now supported by graphic evidence. Indications for the selection of bromides, phenobarbital and hydrantoinates in the treatment of epilepsy and allied states are discussed at some length. The treatment of *petit mal* continues to provide many disappointments and the authors have refrained from reviewing the numerous drugs which have from time to time been in favour. D-glutonic acid hydrochloride is mentioned, but no opinion is expressed about its value.

With regard to the use of sedatives generally in psychiatric practice the warning is repeated against the indiscriminate and prolonged use of bromides for elderly persons. The mental symptoms of bromism are still apt to be overlooked. The administration of chlorides recommended by the authors, in addition to copious fluids, is considered by many undesirable, since too rapid mobilization of bromides in the presence of chlorides may give rise to a troublesome though only temporary aggravation of mental symptoms.

Narcosis therapy, once in favour for the treatment of involutional melancholia, has been superseded by convulsion therapy or insulin coma. But it is still considered useful in the treatment of certain psychoneurotics, provided that there has been no great loss of weight. It may be selected when anxiety or restlessness is a prominent feature in the clinical picture, especially in cases of acute onset. After all, the drowning of sorrows in alcohol is a time-honoured attempt at mental first aid. In spite of modifications of the method introduced by Klaesi some twenty years ago, the treatment has not been freed from risks, especially in the elderly. The authors have omitted to give the formula of Cloetta's sedative mixture which contains seven or eight ingredients. Mental exploration while the subject is under the influence of barbiturates injected intravenously—narco analysis—has proved useful in the acute neuroses of war, for the removal of functional amnesia and of physical conversion symptoms. But, as was the experience in the last war, the removal of an hysterical symptom may be followed by unmasking, release or eruption of a more refractory anxiety state. Again the authors emphasize the importance of dealing with symptoms early in their development. The neurotic of long standing has proved as intractable by this as by other methods. Diet, vitamins and endocrines have a chapter to themselves. The authors discuss the role of vitamin B_1 deficiency in Wernicke's syndrome (hæmorrhagic polioencephalitis occurring in chronic alcoholics), and also refer to deficiency of nicotinic acid in confusional states in elderly people which appear to be precipitated by dietetic upsets and defects in various physical illnesses and after operations. Though physical discomforts may be relieved, the authors are not impressed with the effects of œstrin therapy on the mental state in involutional melancholia. Developments in endocrinology are so rapid that psychiatrists are ever hopeful of something just around the corner which will be of value in their speciality. Frontal leucotomy is being extensively not to say uncritically, performed in some quarters, and a more open operation is advocated than just a trephine and a sweep in the dark. Aggressive and deteriorated schizophrenics may be rendered more tractable and severe "obsessionals" relieved of their mental anguish. The involutional depressives are said to provide some of the best patients for the procedure, but the authors insist that convulsion therapy which is so often successful in this class of case should be given a thorough trial first. The operation leads to some reduction of initiative and self-criticism, its effects are, of course, permanent, and therefore it should be advised only when a certain degree of vegetation appears preferable to otherwise intractable restlessness, aggressiveness or anguish. Like all their confrères in the speciality, Sargant and Slater lament the late application of pyrexial therapy in neurosyphilis which is failing to respond to the conventional treatment. More often the arsenicals are effective after and not before malarial treatment. Inductothermy and the hypertherm apparatus are mentioned, but the technique of malarial therapy only is described. Even pyrexial therapy now seems likely to be superseded by penicillin.

The work ends with a chapter on the relation between psychological and somatic treatment. The authors maintain that the success of physical therapy indicates a decline in the importance of psychogenesis. So often the physician, and still more patients and the laity in general, fail to appreciate when worries are symptomatic or that they may be secondary to difficulties in meeting the stresses and strains of life which themselves arise from a loss of efficiency in the early stages of a mental breakdown. The damaged or fatigued cerebrum becomes unduly susceptible to suggestion and other psychological influences through impairment of the critical faculty. The exigencies of war have encouraged the development of physical therapy in preference to tedious sessions devoted to probing the unconscious. But while physical treatment may be said to be established in the psychoses, it has made only a beginning in the psychoneuroses. The delimitations of these two territories and the selection of appropriate treatment still provide ample scope for nice judgement and wide experience. And physical methods have not relieved the psychiatrist of responsibility for after-care with its manifold psychological problems, for example, rehabilitation including appropriate placement in the social environment. Emphatically these new methods do not mean that one just presses the button and the box does the rest.

Many readers may feel that the authors are overenthusiastic about the success of some of these physical methods, but that is no liability in the practice of a speciality in which active treatment is in process of replacing therapeutic nihilism.

In spite of its modest size, this is one of the most valuable practical works on psychiatry to have appeared in the English language in recent years and deserves close study by every psychiatrist.

"NOTABLE NAMES IN MEDICINE AND SURGERY."

MR. HAMILTON BAILEY, who is known to most of us as a successful author of modern surgical treatises, and Mr. W. J. Bishop, a librarian of the Royal Society of Medicine, have conjointly produced a most interesting and instructive little book entitled "Notable Names in Medicine and Surgery".[1] The method employed in the presentation of this fascinating subject is original in conception, and by reason of the fact that the authors so often lead us pleasantly along unfrequented by-paths of medical history, the book is likely to prove quite a helpful contribution to its study. Few will disagree with the contention expressed in a preface that many proper names are familiar to us and are in everyday use from our medical vocabulary, yet we know little or nothing about the owner of the name or of the circumstances which gave origin to the eponym; for instance, how many of us are conversant with the story behind Lugol's solution, Fowler's position, Colles's fracture, Stenson's duct or the Wassermann reaction?

Each biographical sketch is surmounted with a portrait of the subject and the text is freely interspersed with illustrations of a splint, an instrument, an anatomical dissection displaying the structure first described by its discoverer, the house where he was born or the hospital in which his best work was performed. Many of the names included in this valuable record are omitted from the larger volumes on the history of medicine or are very lightly touched upon, so that the reader has the advantage of learning a number of important facts connected with his work culled from sources not readily accessible to the average research worker. As the authors rightly observe: "The practice of naming a disease after the medical man who first gave a description of it has two advantages. In certain instances it provides a convenient label for a condition which otherwise would be difficult to describe briefly, and it perpetuates the name of the discoverer." The book itself provides a complete justification for these assertions and can be strongly recommended to readers who wish to gain a fuller appreciation of names which are part and parcel of daily practice.

[1] "Notable Names in Medicine and Surgery", by Hamilton Bailey, F.R.C.S. (England), and W. J. Bishop, F.L.A.; 1944. London: H. K. Lewis and Company, Limited. 7¼" × 5", pp. 209, with many illustrations. Price: 15s. net.

The Medical Journal of Australia

SATURDAY, FEBRUARY 3, 1945.

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CUSHING'S SYNDROME.

In 1932 Harvey Cushing wrote[1] that of all the subjects engaging the attention of the medical profession at that time, endocrinology lent itself particularly to the temptation of "impressionistic speculation". This is still true. Cushing, in his original description in 1912 of the syndrome that bears his name and in his subsequent description twenty years later, showed, as he invariably did in his writings, that he knew exactly how far speculation was justified and he did not confuse fact with fancy. When he ascribed his syndrome to basophile adenomata of the pituitary body, he realized that many questions remained unanswered and he expressed the opinion that no doubt an answer would in time be forthcoming to them and to similar questions. The following features were described by Cushing as characteristic of all cases of the syndrome: (*a*) a rapidly acquired, peculiarly disposed and usually painful adiposity, confined to face, neck and trunk, the extremities being spared; (*b*) a tendency to become round shouldered, even to the point of measurable loss of height, associated with lumbo-sacral pain; (*c*) sexual dystrophy shown by early amenorrhœa in females and ultimate functional impotence in males; (*d*) an alteration in normal hirsuities shown by a tendency to hypertrichosis of face and trunk in all females as well as in preadolescent males, and possibly the reverse in adult males; (*e*) a dusky or plethoric appearance of the skin with purplish *lineæ atrophicæ*, particularly marked on the abdomen; (*f*) vascular hypertension; (*g*) a tendency to erythræmia; (*h*) variable backaches, abdominal pains, fatigability and ultimate extreme weakness. Cushing mentioned other features that were "less consistently recorded". That with a syndrome characterized by so many and diverse features speculation has not been excessive is first of all a tribute to the accuracy of Cushing's original observations so far as they went; it also shows that investigators know that they must proceed with care in so difficult a field of investigation. This does not mean that workers have been idle—far from it. At the same time a concise and complete statement on the subject is not readily available. Beattie and Dickson in their textbook state[1] that Cushing first described the condition, and that it was subsequently found to be due to an excess of basophile cells in the pituitary body in the form of an adenoma, and that in addition to the pituitary adenoma, the changes in the organs are those of hypertensive arteriosclerosis. They go on to state that there are no uniformly constant findings, but that hypertrophy of the suprarenal cortex has been observed in some cases, atrophy of the ovaries in others, and rarefaction of bones in several. Many of the findings resemble those produced by suprarenal cortical tumours, and the true nature of the condition may require for its identification the detailed and minute histological examination of the pituitary body. Beattie and Dickson hold that while there seems no doubt as to cause and effect between the basophile adenoma and the clinical syndrome, the exact mechanism is obscure.

The relationship of the suprarenal bodies to the syndrome has given rise to a great deal of discussion and investigation. For example, Grollman states[2] that cases have been reported which were clinically indistinguishable from Cushing's syndrome, but in which at autopsy no disorders of the pituitary body were found. Instead an adrenal tumour was discovered as the possible source of the disorder. In discussing the part played by the adrenals in the production of Cushing's syndrome, Grollman refers to the suggestion of Broster and Vines that all cases of the syndrome may be associated with a change in the adrenals manifested by a different staining reaction of the juxta-medullary tissue. He thinks that if this view was substantiated Cushing's syndrome might be regarded as a manifestation of adrenal virilism, primarily induced perhaps by the pituitary basophilism. It may be, he states, that the pituitary basophilism initiates the observed clinical picture by suppressing the ovarian function which in turn elicits a reaction of the androgenic zone. Since castration in mice also produces an hypertrophy of the androgenic tissue, the disorder might according to this view be caused either by a primary hyperplasia of the androgenic tissue of the adrenal or by a hyperplasia of this tissue induced primarily by a disorder of the pituitary body. Zondek[3] points out that clinically typical cases of the syndrome exist without basophile adenoma of the anterior lobe of the pituitary body. Other tumours have been found to lead to the same syndrome—tumours of the adrenal cortex, certain ovarian tumours and malignant tumours of the thymus. He refers to Bauer's view that the syndrome may be caused by hyperfunction of the adrenal cortex and adds that basophile adenoma would then be but one of several pathogenic possibilities, since with it the adrenal cortex would be excited to enhanced function by a stimulus issuing from the anterior pituitary lobe (primary interrenalism). Cases due to arrhenoblastoma or malignant tumour of the thymus would represent further possibilities of secondary interrenalism. Zondek states that Jones produced hyper-

[1] "Papers Relating to the Pituitary Body, Hypothalamus and Parasympathetic Nervous System", 1932, Chapter 3.

[1] J. M. Beattie and W. E. C. Dickson: "A Textbook of Pathology, General and Special, for the Use of Students and Practitioners", Fourth Edition, 1943, page 815.

[2] A. Grollman: "The Adrenals", 1936, page 353.

[3] H. Zondek: "The Diseases of the Endocrine Glands", Second English Edition, 1944, page 303.

trophy of the adrenal cortex in animals by injecting dialysed serum from six cases of Cushing's syndrome and that the results were confirmed by Anderson and Haymaker. Zondek also mentions observations by Crooke on the basophile cells. Further reference must be made to the work of Crooke, for the basophile cells are obviously of the greatest significance in Cushing's syndrome and his work according to the latest indications is likely to have far-reaching results.[1] Crooke described a hyalinization of the basophile cells of the pituitary body which was present in every one of twelve cases of Cushing's syndrome. He concluded that the change in the basophile cells was an indication of an altered physiological activity which was of fundamental significance in the pathogenesis of the syndrome. In other words the basophile cells showing the hyalinization were the only constantly abnormal feature present. It should perhaps be stated that this hyalinization was found, though only in slight amount, in basophile cells of nine of 350 pituitary glands from various conditions in which the syndrome was not present.

From this short account of the work that has been carried out on Cushing's syndrome it would appear that what is needed most is a determination of the relationship between the adrenal body and the basophile cells of the pituitary. In this regard attention must be drawn to a study on the pathogenesis of Cushing's syndrome that has recently been published by Peter Heinbecker.[2] In the opening portion of his study Heinbecker reminds us that basophile adenomata of the pituitary body may occur without Cushing's syndrome, and also that the syndrome may be unassociated with basophile adenomata. Instead, hyperplasia of the adrenal cortex, a benign or malignant tumour of the adrenal cortex, is the most frequent finding. He explains that confusion existed because of the many typical cases in which no one pathological condition was consistently found. The use of the past tense should be noted here, because Heinbecker believes that the work of Crooke, just mentioned, has settled the question. He states that Rasmussen noted in three cases of clinical basophilism the hyalinization described by Crooke. In one of these a basophilic adenoma was present, in one an adrenal tumour was found, and in the third neither. Rasmussen has, since reporting these three cases, described in a personal communication others in which the hyalinization was present. Heinbecker states that in a search for a primary seat of disturbance leading to the changes found in Cushing's syndrome the hypothalamus, an obvious site, has remained unexplored. It has been shown, he explains, that a properly localized lesion involving the hypothalamic nuclei, particularly the paired paraventricular nucleus, invariably gives rise to obesity. Associated with this condition, produced experimentally in dogs, there have also been found many of the changes in other organs and tissues which are evident at autopsy in persons dying of basophilism. Heinbecker's own report covers both clinical and experimental observations. He gives details of six cases of Cushing's syndrome, in five of which the hypothalamus was available for study. In all of these five cases well-marked hyalinization of the basophile cells of the pituitary was found. In four cases changes were found in the hypothalamic nuclei, particularly the paraventricular nuclei, and in none of these four was an adrenal tumour present. In the fifth case a malignant adrenal tumour was found at autopsy and no hypothalamic lesion was present. In the sixth case of the series an adrenal tumour was removed at operation, but the brain was not available for study. Heinbecker's experimental work was carried out on three dogs. A lesion was produced in the hypothalamus of these animals, the supraoptic and paraventricular nuclei being involved. A diminution in the number of basophile cells of the hypophysis occurred and the remaining cells of this type showed a complete loss of granules, with a homogeneous, turbid appearance of the cytoplasm. The appearance of these cells differed from the hyalinized basophile cells of persons dying of the basophilic syndrome in that the latter cells usually show the hyalin material in direct contact with some remaining normal granular cytoplasm. Heinbecker thinks that the difference in the cytological picture of the basophile cells of his animals and of persons with Cushing's syndrome may depend on a difference in degree of the hypothalamic injury or the difference may be a species difference. He states that the basis for the basophile loss in what he calls his "puncture dogs" has not been established with certainty. We need not follow his discussion on this point, but his conclusion is that possibly an unbalanced or exaggerated influence from the adrenal cortex is responsible. A further observation to be noted is that in Heinbecker's animals there occurred changes in the thyreoid gland, in the gonads and in the islets of the pancreas of a type which serves to explain many of the symptoms of Cushing's syndrome. He also shows that on the basis of his animal experiments the hypothalamic lesions found in his four cases would explain the obesity of Cushing's syndrome. There is no need to follow Heinbecker's discussion on fat metabolism and water balance in the light of his experimental observations, nor need we do more than state his tentative conclusion that the hypothalamic lesion in a case of Cushing's syndrome may be due to a low degree of internal hydrocephalus. What is important is his conclusion that at least three primary lesions, a tumour of the adrenal cortex, a tumour of the thymus or an atrophy of the nuclei of the hypothalamus, particularly the paraventricular nuclei, are the probable precursors of the hyalinization of the basophile cells of the pituitary body as described by Crooke and Rasmussen. In regard to the adrenal bodies, Heinbecker states that the basophile cell change is effected through an unbalanced influence from the adrenal cortex.

This imbalance can be of two kinds; one is excess secretion from the adrenals, the other, normal secretion acting on an hypophysis sensitized to adrenal cortical hormone. The first or excess secretion may be due to hyperplasia or tumor formation in the adrenal gland, the second is illustrated in the case of Cushing's syndrome with paraventricular nuclear atrophy and normal adrenal glands, or in the dog with paraventricular nuclear destruction or denervation and normal adrenal glands.

At this point we may recall that Harvey Cushing in 1932 (*loco citato*) wrote as follows:

. . . if further study should prove that adrenal tumors in the absence of any demonstrable change in the pituitary body may cause a polyglandular syndrome in many respects similar to that under discussion, it may well enough be assumed that, when the same features characterize the syndrome of a basophil adenoma, they in all probability are

[1] A. C. Crooke: "Changes in Basophil Cells of the Pituitary Gland Common to Conditions which Exhibit the Syndrome Attributed to Basophil Adenoma", *The Journal of Pathology and Bacteriology*, Volume XLI, 1935, page 339.

[2] *Medicine*, September, 1944.

secondarily ascribable to a hypersecretory influence of adrenal cortex even in the absence of any histologically appreciable abnormality. . . .

There is no doubt that Heinbecker has carried out a study of extraordinary interest and value. It is rather surprising that so long has elapsed between the publication of Crooke's work and its use by another worker. Perhaps the intervention of the war has been partly responsible. That so long a period will not elapse before Heinbecker's experimental findings are confirmed is earnestly to be hoped. By linking up the basophile cell changes and the adrenal bodies in the suggestive way that he has, he has given at least a logical conception of the whole process. If his ideas should ultimately prove to be correct Cushing's concept of an underlying overactivity of the cells of a basophile adenoma will, as Heinbecker states, become no longer tenable. It might then become necessary to adopt Heinbecker's suggestion that the somewhat high statistical incidence of basophile adenoma found in cases of Cushing's syndrome is indicative of an attempt to compensate for the depression of basophile cell function. But we must hasten slowly and not lay ourselves open to a charge of unjustified "impressionistic speculation".

Current Comment.

INTESTINAL FLORA AND VITAMIN SYNTHESIS.

THAT branch of nutritional study which is classified under the label vitamins displays all too obviously the existence of wide gaps of ignorance which future research, it is hoped, will fill. The exact mechanism of action, the duration of service and ultimate fate, the ability of the body to build up reserves, the possible standardization of concentration in the blood, the loss through sweat and urine, the range of synergism and the possibility that the body's needs may be seasonal like the supply are all topics to which increasing attention is now being paid. One special development of vitamin discovery and one which indicates a new assessment of values in summing up the qualities of certain foodstuffs, is concerned with the synthesis of vitamins by intestinal bacteria and the availability of such endogenous products in the metabolism of the host. The problem why some animals are able to live and thrive on a diet containing only the merest traces of a vitamin regarded as essential was solved when it was demonstrated that the missing vitamin could be elaborated in quantity by intestinal flora. The public has been told repeatedly that inadequacy of vitamin B_1 or thiamin is the most common vitamin deficiency in the diet of civilized man, largely arising from the popularity of white bread. It has been shown, however, that considerable biosynthesis of this accessory factor takes place in the human intestine.[1] Whether the amounts of such thiamin are sufficient, whether the synthesis varies with the food, and whether the bacteria themselves require some thiamin to get going, are questions which research will no doubt answer in the near future. And now we learn that vitamin B_2 or riboflavine can also be formed endogenously in the human intestine.[2] Describing their methods and results in a recent issue of *The Journal of the American Medical Association*, five Baltimore investigators have come to the conclusion "that riboflavin may not be a dietary essential under all conditions". Just what conditions favour the synthesis and what inhibit it, we are not yet told, but information on these points may be expected shortly. A still more recent discovery is that in the laboratory animal Vitamin *K* can be produced by intestinal bacteria and especially by the *Escherichia coli* type, and we need not be surprised if a corresponding synthesis takes place in the human bowel.[1] One fact emerging from this investigation may have considerable significance in the general theory of nutrition, namely, that sulphonamide drugs acting bacteriostatically on the intestinal flora can inhibit this endogenous synthesis and precipitate the animal into vitamin *K* deficiency with consequent hæmorrhages if this factor is not already present in adequate amount in the food.

[1] References to the literature on this topic will be found in the *British Medical Journal* of March 4, 1944, page 332.

[2] V. A. Najjar, G. A. Johns, George C. Medairy, Gertrude Fleischmann and L. Emmett Holt: "The Biosynthesis of Riboflavin in Man", *The Journal of the American Medical Association*, October 7, 1944, page 357.

PULMONARY PHYSIOLOGY AND PULMONARY THERAPY.

THE professor of physiology in the Harvard School of Public Health who delivers a lecture on "The Application of Pulmonary Physiology to Therapeutic Procedures" with special reference to the use of oxygen, is certain to have a well-disposed and attentive audience.[2] Professor C. K. Drinker starts off with a consideration which is well known amongst physiologists, but is worth emphasizing before a clinical gathering. The pressure in the pulmonary capillaries is low and dilatation of their arterioles can never give the increase which is found in systemic capillaries. This low blood pressure has a great practical significance, for in health it is below the osmotic pressure of the blood proteins and hence it cannot bring about a filtration of water and crystalloids from the plasma into the air cells. Drinker speaks of the "dryness" of the air cells, meaning, of course, the absence of liquid exudate. This dryness will be maintained so long as the permeability of capillary wall, with alveolar lining, refuses passage to protein. Should, however, the permeability be increased so that blood albumins and globulins can leak through, then pulmonary œdema will result. Now one of the most important consequences of anoxia is that a pronounced increase of permeability takes place—actually some red corpuscles can slip through as well as protein. This water-logging of the air cells increases the anoxia, and so, in Professor Drinker's words, "anoxia begets anoxia" and a vicious circle is set up. He makes a convincing plea for an intelligent anticipation of anoxia and for the use of oxygen before damage has become manifest. It is a pity that an address with such an engaging title should fall short of the reader's initial expectations. Professor Drinker makes the usual mistake of limiting his attention to the intake of oxygen and neglecting the adjusted liberation of carbon dioxide. The blood leaving the alveoli may display variations in oxygen tension amounting to several parts *per centum* of an atmosphere without any effects on the body generally, but the carbon dioxide tension does not in health show a range of even 1% of an atmosphere. Controlled escape of carbon dioxide is in fact the micro-adjustment of the blood's reaction, but how often does this consideration enter into the mind of the clinician who administers oxygen to his patient by tent or mask? Professor Drinker refers to the lymphatic network, but fails to mention the propulsive action of the massage produced by the rhythmic expansion and contraction of the lung tissue. It is impossible to think that he is ignorant of the pioneer researches of J. S. Haldane. Lastly it should be mentioned that the modern physiologist and pathologist are acutely aware of something else happening to the blood in the lungs than interchange of gases, and it is precisely here that some guidance and suggestions might have been proferred, but these are not hinted at in this address.

[1] *The Journal of the American Medical Association*, September 16, 1944, page 174.

[2] *The New England Journal of Medicine*, October 5, 1944.

Abstracts from Medical Literature.

GYNÆCOLOGY.

Changes in Operative Gynæcology during the Last Twenty-Five Years.

L. E. PHANEUF (*The Journal of the American Medical Association*, September 16, 1944), in an address given recently, summarizes the changes that have taken place in operative gynæcology during the last twenty-five years. Significant improvement in surgical technique has been made, and emphasis has been placed on careful dissection and ligation of individual vessels with fine material rather than on mass ligature of tissues. The treatment of carcinoma of the cervix has changed from surgery to irradiation, with the return to the radical pelvic operation by a few gynæcologists in early cases and "good surgical risks". Carcinoma of the uterine corpus and fundus has remained a surgical lesion, surgical treatment, however, being complemented by irradiation. Improvement in the operation of myomectomy has resulted in more conservative management of these lesions in the young; supravaginal hysterectomy still remains the common method in use, while an increasing number of gynæcologists have turned to panhysterectomy as a prophylactic means against carcinoma of the cervical stump. Vaginal hysterectomy has been reborn and improved and has now become a commonplace procedure. The increased number of surgical vesico-vaginal fistulæ has been responsible for the elaboration of new techniques in the cure of this lesion. Trachelorrhaphy and amputation of the cervix are less frequently performed, having in many cases been replaced by cauterization and electrocoagulation. A significant advance has been made in the surgical treatment of uterine prolapse, cystocele and rectocele through better anatomical understanding of these lesions, and by the reconstruction of the deficient supports through the vagina rather than by a dependence on abdominal suspension or fixation of the uterus. Pelvic inflammatory disease has been handled more and more by conservative methods, and the sulphonamide drugs seem to show great promise in lessening and eradicating this condition. Ovarian tumours have been better classified, the rare tumours have been discovered, the tendency to malignant changes in these neoplasms has been emphasized, and their early ablation has been strongly advised.

Zondek's Simplified Treatment of Secondary Amenorrhœa.

RITA S. FINKLER (*The American Journal of Obstetrics and Gynecology*, July, 1944) draws attention to Zondek's treatment of secondary amenorrhœa and reports its use in a series of 31 patients. The method consists of injection on two consecutive days of 2·5 milligrammes of œstradiol benzoate and 12·5 milligrammes of progesterone. These substances were drawn into the one syringe and injected intragluteally. In this series bleeding was found to result in 80% of cases. It occurred in from four to six days following the second injection. In some instances two courses of therapy were given. In general it was found that the treatment was most suitable for cases of amenorrhœa of less than two years' duration. In only four of the 31 cases did more than one bleeding episode follow the treatment, except when other therapeutic measures were employed. It was found that the two-day hormonal treatment did not produce a secretory pattern in the endometrial mucosa, in spite of its efficacy in producing bleeding. However, the treatment is considered valuable in those cases of secondary amenorrhœa due to hormonal disturbances, in which the condition appears to produce depression or neurosis.

The Blood Phosphates in Obstetrics and Gynæcology.

SADOWSKY (*Revue française du Moyen-Orient*, May, 1944) has investigated the blood phosphates, with special reference to their role in obstetrics and gynæcological conditions. His observations are based upon a series of 100 cases; some of the subjects were normal, non-pregnant women, and others were pregnant, or had been delivered, or were suffering from various disorders. The author found that the normal limits of phosphate content in the blood varied widely; he concluded that, in spite of the fact that in certain disorders (toxæmia of pregnancy, post-partum fever, parametritis *et cetera*) the action of phosphatase is increased, it is impossible to draw diagnostic conclusions from the action of the blood phosphatase. He agrees with Vermehren that the amount of phosphatase in the blood is not increased at the commencement of pregnancy; this observation holds both for the phosphatase of glycerophosphoric acid and for the phosphatase of inosinic acid. In general it may be stated that no disturbance of phosphate metabolism takes place during pregnancy.

The Control of Menorrhagia by Prolactin.

HERBERT KUPPERMAN, PAUL FRIED AND L. Q. HAIR (*The American Journal of Obstetrics and Gynecology*, August, 1944) have investigated the action of prolactin in controlling excessive uterine hæmorrhage and also in a few cases of threatened abortion. The rationale for this therapy is based on the fact that the only true period of physiological amenorrhœa other than pregnancy occurs during the interval of lactation. Also experimental evidence has demonstrated the antigonadal effect of lactogenic preparations. Very successful clinical results were obtained in controlling menorrhagia of both functional and organic origin. Patients suffering from the latter group, including those suffering from *fibromyoma uteri*, ovarian cysts *et cetera*, were more resistant to therapy, but it was found that a course of treatment with lactogenic hormone was very useful as a preliminary to surgery. Administration to normal cyclic women was found to have no appreciable effect upon the length of the cycle, menstrual bleeding or morphological appearance of the endometrium. Endometrial biopsies taken from the menorrhagic patients showed that the prolactin therapy did not bring about any change in the morphological appearance of the menstrual endometrium, even though it successfully controlled the excessive uterine bleeding. As many of the endometrial patterns were predominantly œstrogenic or proliferative both before and after treatment, it seems obvious that the beneficial effect of the prolactin was not due to its antigonadal properties. The authors favour more the theory that it operates through its powerful luteotrophic action, both in controlling menorrhagia and in holding threatened abortion in abeyance.

Primary Masculinizing Tumours of the Ovary.

J. A. BURKET AND I. ABELL (*Surgery, Gynecology and Obstetrics*, December, 1944) discuss primary masculinizing tumours of the ovary. The two tumours of this variety are the arrhenoblastoma and the adrenal cell tumour, and it is almost impossible to make a clinical distinction between them. Edgar Norris concluded that the term arrhenoblastoma should be used only when both the clinical and pathological observations justified its use; that is to say, it is a clinico-pathological condition. The term should be reserved for those cases in which there are definite evidences of defeminization and possibly masculinization associated with an ovarian tumour which can be so classified microscopically. Thus it is impossible to make the diagnosis on the microscopic appearance alone. This tumour is uncommon. In 1938 Novak reported that there were 45 cases of arrhenoblastoma and added six others to the list. Norris in a critical study excluded cases in which virilism was not present, and reduced the number to 29. The authors have been able to find 20 additional cases. The adrenal cell tumour of the ovary is quite rare. The authors find in the literature records of 15 cases and they report one of their own. The patient was a woman, aged thirty-six years, who was admitted to hospital complaining of an abdominal tumour which was first noticed after dieting and losing 40 pounds of weight in the previous year. Questioning revealed that she had not menstruated since the birth of her second child sixteen years previously. Shortly after the birth of her child hirsutism began to develop, the face and neck became flushed and bluish and the voice deepened. At operation numerous metastases were found in the liver and subsequent coma suggested cerebral involvement. Nine days after the operation the patient menstruated for the first time in sixteen years. The patient died on the eleventh day after operation. Permission for autopsy was refused. Histological examination of the tumour showed that it was of adrenal cortical cell origin, primary in the ovary.

Adenocarcinoma of the Ovary.

OTTO SAPHIR AND J. E. LACKNER (*Surgery, Gynecology and Obstetrics*, November, 1944) state that there is probably no organ within the body in which so many distinct tumour entities are recognized as in the ovary. They report two yellow, malignant tumours of the ovary which, they hold, were in no way related to those producing masculinization. These tumours were identical with carcinomata occurring in the kidney. Several pathologists, not knowing that sections of the tumour were from an ovary, diagnosed a kidney

tumour. The kidney tumours which these tumours resembled are classified as hypernephroid carcinoma and are sometimes referred to as renal carcinoma, adenocarcinoma with clear cells or malignant nephroma. The tumour is characterized by a yellow colour and evidence of necrosis and hæmorrhage. Histologically the outstanding features are large cuboidal or polyhedral cells which may contain fat or glycogen. There is not on record a single recognized instance in which this tumour has caused changes in the secondary sex characteristics. It arises apparently from mesonephric structures. This tumour must be distinguished from another type of yellow ovarian tumour. This type causes masculinization and is either a luteinized granulosa cell tumour and may be called lutein cell tumour, luteinoma *et cetera*, or if it arises from suprarenal cortical structures it is called hypernephroma.

OBSTETRICS.

Cæsarean Section.

CLARENCE C. BRISCOE (*The American Journal of Obstetrics and Gynecology*, July, 1944), in reviewing the results of 409 Cæsarean sections, comes to the following conclusions: (i) Cæsarean section performed before labour begins offers the patient the most protection against sepsis. (ii) Post-Cæsarean morbidity rates at the Philadelphia Lying-in Hospital during the past three years show superiority of the low section over the classical section for the elective case and especially for the patient in labour. (iii) Over a third of the women who die in Philadelphia following Cæsarean section die of sepsis. (iv) One-half of these deaths occurred among patients who had been in labour over twelve hours before operation, 37% of whom previously showed signs of sepsis. (v) The absolute and the relative death rates from sepsis show superiority of the low operation for the elective case and especially for the patient in labour. (vi) The supposed protection of the Porro operation for the infected patient has been lacking in Philadelphia during the past twelve years. (vii) Reported series of extraperitoneal operations show the death rate from all causes to be from two to five times less than the death rate from sepsis alone for the Porro operations. (viii) The operation of choice for the infected patient would seem to be an extraperitoneal Cæsarean section.

The Infant at Birth and the Basal Metabolism of the Mother in Pregnancy.

L. W. SOUTAG, E. L. REYNOLDS AND V. TORBET (*The American Journal of Obstetrics and Gynecology*, August, 1944) study the relationship between a woman's basal metabolic rate and the birth weight of her infant. The data for this study were obtained by estimating the basal metabolic rate in the ninth month of pregnancy and the gain in basal metabolic rate from the beginning of pregnancy to the ninth month. Infants of mothers with high basal metabolic rates at the ninth month of pregnancy were found to be heavier and longer at birth and more advanced skeletally at the age of one month than infants of mothers with low basal metabolic rates. However, the gain in weight, height and ossification during the first six months was greater in the latter group. The mothers who showed a high basal metabolic rate in the ninth month were also in many cases the mothers who showed a high gain in basal metabolic rate from the beginning of pregnancy and *vice versa*. The findings in regard to the infants corresponded closely in both cases. It is pointed out that these findings appear to be contrary to those of observers who have claimed to reduce fœtal obesity by feeding pregnant women three to six grains daily of desiccated thyreoid gland. It is pointed out, too, that the effect of hyperthyreoidism on young animals is retardation of growth. Although the pregnant woman may be tolerant of these large doses of thyreoid gland because of the development of antihormones or the inhibitory action of other endocrines, the fœtus probably has no such protection. The high blood levels of thyreoid hormone that such treatment involves might induce in the fœtus a state of hyperthyreoidism sufficient to depress growth. Therefore such treatment is not recommended for normal women. If, however, the mother has a deficiency of thyreoid, the raising of the hormone level to its optimum state would enable the full exercise of its usual physiological function of promoting growth.

Spontaneous Annular Detachment of the Cervix during Labour.

M. D. WESTERMAN (*Proceedings of the Royal Society of Medicine*, June, 1944) reports a case of spontaneous detachment of the cervix during labour. The patient was a healthy *primigravida*, aged twenty-seven years. She had a flat type of pelvis. She was booked for trial labour. Two weeks before the expected date of delivery labour commenced following spontaneous rupture of the membranes. The vertex was in the right occipitoposterior position and very high. The patient was uncooperative. The first stage of labour lasted forty-three hours and the only abnormal sign was a rise in the pulse rate. After an hour of poor pains in the second stage no progress had been made and it was decided to apply forceps. During rotation of the head a loose band of œdematous tissue was felt between the head and the sacrum. This was found to be the prolapsed anterior cervical lip; the cervix having sloughed round the anterior two-thirds of its circumference, the head was presenting through the rent. As the vital part of the cervix had separated, it was thought that the danger of hæmorrhage from tearing of the vaginal vault was over. Forceps were applied and the detached cervix came away with the head. On the patient's discharge from hospital a smooth regular cervical stump was palpated.

Herpes Gestationis.

CHARLES W. MUELLER AND WARREN A. LAPP (*The American Journal of Obstetrics and Gynecology*, August, 1944) describe *herpes gestationis* as a chronic polymorphous exanthem characterized by recurrent crops of bullæ and vesicles on erythematous bases and associated with severe pruritus. The ætiology is unknown, although many theories have been advanced. Many authors believe that the lesions result from injury to the vasomotor nerves by toxins or ferments manufactured by the fœtal tissues or by cells of the chorionic epithelium. Others, however, maintain that hormonal factors are responsible, while allergic phenomena to various substances have been blamed. The disease commonly appears in the latter half of pregnancy. It tends to occur with successive gestations. Once the disease appears it usually progresses to the termination of the pregnancy, showing many cyclic exacerbations and remissions. During the first few days *post partum* a final acute flare-up is usually followed by a permanent regression of all signs within two weeks to three months. The pruritus associated with the disease is intense and persistent. The prognosis for the mother is fairly good, but for the child it is less hopeful. The incidence of spontaneous abortions, monstrosities and stillbirths is high. Cutaneous lesions may occur in the child. The treatment suggested is very varied, but in the majority of cases the course of the disease seems to be little influenced by the therapy. The prevention of secondary infection and general supportive measures are the most important. Autohæmotherapy, serum from normal pregnant women and injections of horse serum have been tried with varying success. Other workers recommend Fowler's solution, hormonal treatment or sulphonamide drugs. In severe cases, interruption of the pregnancy is advised by many.

Eclampsia without Convulsions, Hypertension or Coma.

RALPH A. REIS AND ELI A. BERNIES (*The American Journal of Obstetrics and Gynecology*, August, 1944) review the literature of eclampsia without convulsions or coma and report such a case from their own practice. The patient was a *primigravida*, aged twenty-nine years, and on her first examination at fourteen weeks had a blood pressure of 110 millimetres of mercury, systolic, and 80 millimetres, diastolic, and normal urine. Subsequently the blood pressure gradually rose, until at twenty-eight weeks it was 145 millimetres of mercury, systolic, and 90 millimetres, diastolic. With rest in bed it subsided to 130 millimetres of mercury, systolic, and 80, diastolic, but the urine began to contain albumin. It was noted at this time that the uterus appeared to be decreasing gradually in size, although the fœtal heart sounds remained good. While undergoing treatment with bed rest and the usual diet, the patient complained of mild epigastric pain, which, in the space of a few hours, became gradually more severe, so as to simulate biliary colic. The blood pressure was 128 millimetres of mercury, systolic, and 86 millimetres, diastolic, no œdema was present and the patient was fully alert. Soon afterwards she appeared to pass into a faint, which led to death. *Post mortem* the liver was found to be enlarged and to contain large areas of hæmorrhage, associated with necrosis of the liver tissue. There were petechial hæmorrhages in the endocardium and cloudy swelling of the myocardium and kidneys. The placenta had several large infarcts and the fœtal sac was completely devoid of amniotic fluid. The authors suggest a possible relationship between this absence of amniotic fluid and the toxæmia.

British Medical Association News.

THE OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA (BRITISH MEDICAL ASSOCIATION).

ANNUAL MEETING.

THE fourth annual general and scientific meeting of the Ophthalmological Society of Australia (British Medical Association) was held at the lecture hall of the Royal Australasian College of Surgeons, Spring Street, Melbourne, on October 12, 13 and 14, 1944, DR. G. H. BARHAM BLACK, the Vice-President, in the chair. Those present included one member from Queensland, fifteen members from New South Wales, twenty-nine from Victoria, two from Tasmania and four from South Australia.

Vice-President's Address.

The Vice-President, Dr. G. H. Barham Black, extended the welcome of the society to such guests as had been able to attend, and noted with pleasure the presence of three former presidents of the society, Sir James Barrett, Surgeon Commander A. James Flynn and Dr. Leonard J. C. Mitchell. Dr. Barham Black paid tribute to the late president, Dr. J. Lockhart Gibson, of Brisbane, whose recent death had deprived the society of a distinguished member and an esteemed leader. Dr. Barham Black referred to the interval of three years which had elapsed since the last annual meeting of the society, owing to war conditions, and then delivered an address which dealt with various aspects of ophthalmology. He also indicated that the part of his address dealing with penicillin in its application to ophthalmic practice would be given at a later session of the meeting when this subject came up for discussion.

Dr. Barham Black then referred to a new type of disease which had been termed kerato-conjunctivitis, and described its signs, its symptoms and its differences from somewhat similar conditions previously known, for example, superficial punctate keratitis. He summarized his remarks by saying that there was a very considerable group of diseases of the cornea, the essential signs of which were punctate, epithelial or sub-epithelial lesions, the classification of which was confused, the ætiology uncertain and the treatment unsatisfactory.

Dealing with the industrial aspect of ophthalmology, Dr. Barham Black said that he had long felt that the society should take definite steps in advising various interested bodies on measures which could and should be adopted to prevent the grave economic wastage arising from preventible ocular damage. He described the principal industrial operations from which such accidents arose, in particular the use of abrasive wheels and other high-speed machinery, and expressed the opinion that the compulsory use of protective devices could prevent a large percentage, estimated by some observers as being as high as 98%, of such accidents. Lack of proper or sufficient illumination led to conditions which could be considered under two headings, subjective symptoms (fatigue and eye-strain on the part of employees) and objective results (loss of efficiency and failing output on the production side). He indicated that ophthalmologists had to deal with patients exhibiting the subjective symptoms, and were seriously handicapped by their inability to remedy the cause which all too frequently was the lack of proper lighting. Conversely he realized that the illumination engineers were also handicapped in the provision of adequate lighting owing to the absence of authoritative medical opinion on the varying needs of lighting for particular tasks. Moreover, there were innumerable instances of factories and workshops in which expert advice on lighting needs was never sought, and in such places the problem was aggravated. This criticism applied equally to schools, banks, offices, churches, and to most establishments in which precision work was controlled by vision. He expressed the view that the society should endeavour to focus the attention of all interested bodies upon this problem.

Dr. Barham Black said that he would defer the portion of his address dealing with post-graduate ophthalmic education until a later session of the meeting, when he would open the discussion on this subject. He expressed the wish that, contrary to accepted custom, any or all of the subjects so far dealt with in his address should be open to general discussion.

Eye Injuries in Industry.

DR. E. COUPER BLACK (Adelaide) read a paper dealing with the commoner eye injuries occurring among about 13,000 employees of the South Australian Railways, including those engaged in workshops, on trains and as labourers. About three-quarters of the injuries were among the workshop people, who formed a little under one-half of the total employees. The numbers of eye injuries reported in the years 1942-1943 and 1943-1944 were 1,512 and 967 respectively. They were divided into three classes; 71% were of a trivial nature involving simple treatment and no loss of time, 29% were cases of foreign bodies embedded in the cornea, involving a mean loss per case of 4·6 days with a total compensation payment of £751 for the year, and a small number (ten over a four-year period) came into the class of serious damage to the eye from penetrating foreign bodies. The working time lost by nine of the subjects with serious sequelæ amounted to 800 days, with a total compensation payment to date of £2,440. As in all but one case the damage happened at hammering, the danger of this procedure was stressed. The causes of the eye injuries were chiefly small flying particles associated with the various occupations. Methods of prevention were discussed, emphasis being laid on the bringing about of a consciousness of the danger; the merits and demerits of goggles were gone into, and the present methods of using screens, a flow of water and suction were described.

With regard to locomotives, Dr. Black said that as engines became larger and faster and burned more coal, flying cinders became greater hazards. Designers were bringing about improvements in dealing with the smoke, and better protection was afforded by closed cabs with large windows in front to obviate enginemen's driving with their heads out at the side. Dr. Black's paper was illustrated with photographs of workshop equipment and locomotive cabs.

Eyesight Conservation and Lighting: The Need for Medical Interest and Leadership.

MR. L. D. WRIGHT, A.M.I.E. (Australia), referred to the unsatisfactory seeing conditions which had existed in Australian industry in 1941, when he presented to the society his paper entitled "Modern Trends in Illumination", and traced the history of the great improvement which had since resulted from the wide introduction of proper standards of illumination. In this regard, the yardstick used was the Australian standard code for the interior illumination of buildings, which had been applied by Commonwealth regulations to all new or altered installations in private and government factories engaged on essential wartime work. The basis of the standards of this code was explained, and reference made to some of the technical problems associated with its application, particularly in the matter of the brightness contrasts in industry essential for comfortable seeing. Experience had shown that the provision of satisfactory brightness contrast conditions went beyond the design of the lighting installations, and involved the colour and brightness of the machines, the work surfaces and the whole environment.

Reference was made to the lack of natural lighting standards, resulting in inadequate natural lighting in many modern industrial buildings, particularly those based on the minimum of the widely accepted rule that light-transmitting area should be equal to at least 10% of the floor area. Work had, however, commenced on an Australian standard code for the natural lighting of buildings, and it was held that the results of extensive researches of the committee established in England by the Ministry of Scientific and Industrial Research should provide valuable guidance.

Passing to the main theme of the paper, Mr. Wright described the unsatisfactory conditions resulting from lack of concerted effort towards creating and popularizing those standards and practices usually classified as eyesight conservation, in which natural and artificial lighting played an important part. The needs of industry, schools, houses and streets were discussed and a description was given of the organization of the Eyesight Conservation Councils of United States of America and Canada. Mr. Wright said that in industry and schools particularly, there was a need for extensive eyesight surveys and associated correction, whilst the resultant statistics of age and occupational groups would be valuable. Not only would correction of eye defects improve the efficiency and well-being of the workers, but it would also advance visual safety, which was a matter requiring attention, particularly regarding the policy and practice of the use of goggles for protection both from flying particles and from injurious rays. The development of safe practices regarding the use of hot or corrosive liquids, proper lighting and colouring of hazards, and other matters of visual safety also needed attention. Colour was widely and badly used in industry and invariably selected from maintenance or æsthetic viewpoints, whereas, used in accordance

with scientific "seeing" principles, attention being paid to psychological and æsthetic considerations, colour could have a great influence on the seeing conditions in industrial, school and other buildings. In conclusion, Mr. Wright stressed the need for proper lighting standards in the post-war community and pointed out the need for medical guidance and leadership in the wider field of eyesight conservation, as other professions were interested only in special aspects and in any case lacked physiological and scientific knowledge of vision.

Lighting and Seeing.

DR. A. L. TOSTEVIN (Adelaide) gave an historical survey showing how nature had been caught unawares by the sudden transition brought about by the introduction of electric lighting in the last sixty-five years, and how little had been done by ophthalmologists to help in the scientific application of lighting principles. Illumination was discussed under the headings of (i) general light, (ii) local lighting, (iii) lighting for highly specialized tasks and (iv) maintenance of lighting equipment.

Emphasis was then laid upon the important question of visual acuity. The term "eyesight conservation" was criticized as being physiologically unsound, and the term "eyesight improvement" was suggested to replace it. Stress was laid upon the necessity for visual surveys as soon as medical personnel became available, and it was pointed out that this question would almost certainly form an important part in any national health scheme that might be evolved by the Federal Government.

In conclusion, Dr. Tostevin made the following suggestions:

1. That members of the Ophthalmological Society of Australia (British Medical Association) join the Illumination Engineering Society of Australia and take an active interest in its work and meetings. The association would be mutually beneficial.

2. That the society form an "eyesight improvement" council with representatives from each State who could meet members of a similar council composed of representatives from illuminating engineers and other interested professional and technical bodies. In this connexion Dr. Tostevin added that if the Federal Government acted in the matter, its appointees could join such a council. They had a precedent for this in Canada and America, where many so-called "eyesight conservation councils" were functioning in an active manner and where the medical profession was playing an extremely prominent part.

Dr. Tostevin then gave the following summary in the form of a *questionnaire*:

1. Is illumination adequate for safe and easy seeing?
2. Is direct or reflected glare interfering with the worker's efficiency?
3. Are shadows handicapping the worker and creating accident hazards?
4. Are the surroundings contributing the best possible seeing conditions?
5. Is the maximum efficiency of the lighting system ensured by adequate and continuous maintenance?
6. Is everything being done to protect the worker's eyes?
7. Is a sight survey possible?

Protective Glass for Welding.

SURGEON COMMANDER A. JAMES FLYNN (Sydney) submitted a draft Australian specification for a protective glass for welding with which he had been concerned as a coopted member of the subcommittee of the Standards Association of Australia. He described the specification and asked for members' criticism and advice.

Tropical Ophthalmology.

CAPTAIN WEBB CHAMBERLAIN (United States Army) opened a discussion upon tropical ophthalmology and spoke extensively on several eye conditions which he had observed during his service in the South-West Pacific Area.

CHAPLAIN FRANK FLYNN read a paper entitled "Excessive Drying Suggested as a Factor in the Ætiology of Conjunctival Hyperæmia". He stated the incidence of conjunctival hyperæmia among convoy drivers in the Northern Territory. He presented meteorological graphs of the evaporation rate for winter and summer and showed their correlation with the incidence of conjunctival hyperæmia. Susceptibility to the condition was determined by certain subjective factors: the rate of lachrymation, the width of the palpebral fissure, the prominence of the globe and the frequency of blinking. The hyperæmia involved the bulbar more than the palpebral conjunctiva and appeared as two triangular areas, base towards the limbus on either side of the cornea. Chaplain Flynn suggested that the condition when recurrent was associated with the development of pterygia. In a few cases corneal involvement occurred in association with the conjunctival hyperæmia, and took the form of superficial punctate lesions confined to a horizontal band across the cornea corresponding to the area exposed by the lids when contracted to a slit. Chaplain Flynn recommended the service eyeshields anti-gas MK2 as a simple and efficient protective device.

A paper by MAJOR K. B. ARMSTRONG (New South Wales) was read in his absence by CAPTAIN M. STERLING-LEVIS. Major Armstrong based his communication on his ten months' experience with a hospital in New Guinea. Much of the palpebral and conjunctival trouble he had met was the result of incessant sweating, lack of ordinary cleanliness, allergy and dandruff. He had found trouble in the treatment of infections due to hæmolytic *Staphylococcus aureus*, coagulase-positive, until he used penicillin.

MAJOR CLIFFORD COLVIN (New South Wales) said that to cover the whole field of tropical ophthalmology would be a colossal task and beyond the scope of his paper. He proposed to deal with a few specific diseases of which he had had some experience.

Dealing first with malaria, Major Colvin said that Bothman had stated that the eye complications which accompanied or followed malaria included everything from lachrymation to optic atrophy. Major Colvin thought the percentage of ocular manifestations was much less than ten, as suggested by Poncet. In his experience conjunctivitis, dendritic ulcer, hyperæmia of the disk, a mild optic neuritis and small retinal hæmorrhages were the ocular signs seen in malaria; ocular pain or headache was a prodromal symptom of the disease.

Major Colvin went on to say that scrub typhus was a disease of rickettsial type. The effects of the disease were caused by small petechial hæmorrhages from the capillaries. The ocular complications were those produced by such lesions. Iritis might occur, also vitreous floaters. In three cases out of 100 fundus changes were noticed about the third or fourth week; in one case only did permanent blindness and deafness occur. All other fundi eventually became normal, though some enlargement of the blind spot was found.

Major Colvin said that the onset of dengue fever was characterized by severe headache and acute pain in the eyes. No lasting effects were noted, but asthenopia might persist for some time. Sandfly fever produced similar symptoms, but no lasting defects. Relapsing fever was associated with facial paralysis and resulting ocular effects with late onset of iridocyclitis. In dysentery, conjunctivitis, iritis and iridocyclitis might occur. Amœbic dysentery might cause chorioiditis. Major Colvin said that he had encountered one case of leprosy with corneal leproma. Bacilli were found in the tumour when it was excised. The patient lived till the age of seventy-four years, and had never been outside Australia.

DR. JOHN POCKLEY (Sydney) described three conditions seen at an Australian general hospital in New Guinea between August, 1942, and September, 1943. The first condition was dendritic corneal ulcer, of which 78 cases had occurred; all the lesions were unilateral. Dr. Pockley emphasized that the conjunctival changes preceded the corneal ulcers and were a better guide to recovery than the corneal signs. The ætiology was discussed, and it was shown that in this series a variety of factors operated to precipitate the disease, malaria, starvation and scrub typhus being the main factors. The association between dendritic ulcer and malaria was seen in less than half the cases. Many degrees of severity of this type of *herpes simplex* infection were encountered, and in some cases the lesion simulated trachoma in the developed stages. Daily brushings with silver nitrate (2% strength) controlled the great majority of ulcers; other forms of therapy having no effect were detailed. The greatest incidence of the disease was observed amongst troops fighting along the Kokoda trail and amongst the heavily disease-infected troops of the Buna-Gona battles.

The second condition discussed by Dr. Pockley was scrub typhus and its eye complications. Thromboses in the central retinal vessels, sub-conjunctival hæmorrhages and paralysis of accommodation in the post-convalescent period were the

main ocular complications of the disease. In approximately 600 cases of scrub typhus, seven eyes with some degree of retinal arterial thrombosis were seen; this varied from one complete bilateral retinal arterial thrombosis to a small partial thrombosis of one branch artery. Two patients who showed such a vascular accident did not survive. Paralysis of accommodation in convalescence was common and troublesome; it lasted for weeks or months if tropical service continued. Finally Dr. Pockley discussed quinine amblyopia. He said that the conditions of malaria control and treatment were by no means ideal in the latter part of 1942. Massive and uncontrolled doses of quinine were often being taken, and always the dosage of quinine was high, especially for the malignant and cerebral forms of malaria. However, only one case of quinine amblyopia was met with, and this in an American soldier who had taken a course of quinine of astronomical proportions. The risk of quinine amblyopia in soldiers under ordinary therapy could be taken to be very low indeed.

Tuberculous Eye Lesions in Immune, Desensitized and Sensitized Patients.

DR. J. BRUCE HAMILTON (Hobart) followed the work of Wood and Burky of the Wilmer Institute reported in *Archives of Ophthalmology* from 1938 to 1943, and analysed the case records of twelve patients who had lived in contact with relatives with active and open tuberculous lesions. Dr. Hamilton showed that the eye lesions these patients developed were heterochromic cyclitis, Eale's disease or phlyctenulosis. He assumed that the two former diseases occurred in persons immune and desensitized to tuberculosis, while the last-mentioned disease attacked persons immune but sensitive to tuberculosis. Dr. Hamilton stressed the fact that the all-important treatment factor in these three diseases was to remove the patient permanently from his infected relative.

Some Observations on Occlusion and Stimulation in the Treatment of Amblyopia ex Anopsia.

MISS L. GILCHRIST (Hobart) recommended partial instead of total occlusion in the treatment of *amblyopia ex anopsia*, in order to ensure the maximum cooperation of the ambliyopic child and its parent. This was combined with daily classes for visual stimulation. By this means the risk of street and home accidents to the child was removed, physiological reactions were reduced to a minimum and education could be continued without complete interruption. Cellulose tape was recommended as the ideal medium for occlusion.

Ocular Tension Relative to Diseases of the Cornea.

DR. A. L. TOSTEVIN (Adelaide) drew attention to the fact that to lower even normal ocular tension by the trephine operation allowed a greater blood supply to the cornea, which caused a favourable response in many diseases of the cornea that did not respond to conservative treatment. These diseases included corneal ulcer, bullous keratitis, *herpes ophthalmicus*, radiation keratitis, conical cornea and kerato-conjunctivitis.

Infantile Dacryocystitis.

DR. J. L. R. CARTER (Launceston) reported a series of 69 cases of infantile dacryocystitis seen in private and hospital practice during a period of three years. Seventeen patients had the condition bilaterally. Of the unilateral cases, in 34 the right eye was affected and in 19 the left eye. Age varied from one week to two and a half years. All the patients were treated under general anæsthesia by dilatation of the punctum and passage of a Bowman's probe into the nose, and then boric lotion was syringed through after withdrawal of the probe. Seven patients required repetition of the dilatation, probing and syringing. Apart from this, there were no complications and no further treatment was required.

Congenital Cataract in Children following Maternal Rubella.

DR. N. MCALISTER GREGG (Sydney) reviewed further developments which had taken place, both at home and abroad, in the investigation of congenital cataract in children following maternal rubella. Mention was made of the important contributions in THE MEDICAL JOURNAL OF AUSTRALIA in 1943 from the Institute of Medical and Veterinary Science, Adelaide, by Swan, Tostevin, Moore, Barham Black, Mayo and Evans. As additional congenital defects occurring in these children they had described deaf-mutism, with or without cardiac disease, cardiac disease alone, microcephaly, and dental defects. A short review was presented of ninety additional cases from New South Wales, from returns made in answer to a *questionnaire* issued by the Department of Public Health; deaf-mutism was the most frequent defect noted in this series. Dr. Gregg said that two committees had been formed in New South Wales. One formed by the Department of Public Health would deal with all aspects of the problem; a second convened by the Department of Public Instruction would investigate the training of the affected children. Reference was made to articles on the subject from *The Lancet* in England and to a contribution in the *American Journal of Ophthalmology* by A. B. Reese. An extract was included from a paper by D. G. Carruthers on thirteen cases of deaf-mutism, and a summary by D. G. Vickery of fourteen cases of mental backwardness in children following maternal rubella.

Dr. Gregg then dealt with a further series of eight cases in babies born in 1943. In seven of these a history of maternal rubella was obtained. The defects noted included cataract, microphthalmos, cardiac disease, deaf-mutism, buphthalmos, mental retardation and dental defects. Finally attention was drawn to the necessity for further investigation into the question of congenital defects in general.

DR. CHARLES SWAN (Adelaide) was prevented by illness from attending the meeting, and his paper was read by Dr. A. L. Tostevin. Dr. Swan summarized the results of research work performed in South Australia by A. L. Tostevin, the late Brian Moore, Helen Mayo, G. H. Barham Black, Mervyn Evans, D.D.Sc., and himself. The following results and conclusions, based on a study of 70 cases of rubella during pregnancy, of which 49 resulted in congenital abnormalities, were presented. Although on clinical grounds there was little doubt that the disease was rubella, conclusive proof must rest upon the isolation from the mothers at the time of the infection of a virus identical with that of German measles. Dr. Swan stated that confirmatory experiments in pregnant susceptible animals should also be attempted. In view of the universal occurrence of German measles, it seemed curious that, if congenital defects at all often followed a maternal attack during pregnancy, the connexion between the two conditions was not discovered earlier. It was postulated that the virus had altered in virulence or had undergone some more subtle change within the last few years. The mutation might be the result of the abnormal conditions occurring in wartime. In the 49 cases in which babies were congenitally malformed, 46 of the mothers had contracted rubella within the first four months of pregnancy; in 12 cases the disease occurred in the first month, in 22 in the second, in nine in the third and in three in the fourth. Other investigators had noted that avian and other mammalian embryos showed, in contrast with adult tissues, an increased susceptibility to infectious agents. It seemed logical to assume that the human embryo possessed a similar enhanced susceptibility to infection.

Dr. Swan went on to state that the average birth weight of 45 of the babies with congenital defects was five pounds eleven ounces, and that of sixteen babies without apparent abnormality was six pounds twelve and three-quarter ounces. There were twenty subjects (nineteen babies and a three months' fœtus) with eye defects. The abnormalities included seventeen cases of cataract, one of buphthalmos, one of bilateral gross central visual defect and one of squint. Of the seventeen babies with cataract, ten suffered also from heart disease, three were mentally deficient and one suffered also from *talipes equino-varus*. There were fifteen cases of deaf-mutism; five of the babies (one-third of the subjects) suffered also from heart disease. Heart disease was present in over half of the cases of cataract. No definite conclusions could be drawn from such a small number of cases, but it appeared not improbable that cardiac abnormalities occurred more often in association with cataract than with deaf-mutism. For the most part the patients were not totally deaf. The majority could hear high-pitched sounds. Speech (when present) was limited to a few words.

Dr. Swan went on to state that at the onset of the exanthem the average duration of pregnancy of fourteen of the fifteen mothers whose children suffered from deaf-mutism was two to three months. The average duration of pregnancy under similar conditions of the seventeen mothers whose offspring had cataract was one to four months. It appeared, therefore, that the nature of the congenital defect depended upon the stage of pregnancy at which the mother contracted rubella. Cardiac abnormalities were present in 26 cases of the series. Seven of the children suffered from heart disease without other apparent defect, ten suffered from cataract, five from

deaf-mutism, one from buphthalmos, two from mongolism and one from squint. Microcephaly occurred in 39 cases; it was pronounced in sixteen, moderate in seventeen and slight in six. In two cases this was the only defect noted. In at least four cases the abnormality was associated with mental retardation. In a few instances, owing to prematurity, microcephaly might have been apparent rather than real; when it was slight it was probably not significant. Other malformations observed included one case of hypospadias, one of obliteration of the bile ducts and two of mongolism. In the absence of further cases, the possibility could not be denied that the occurrence of these defects in association with rubella in pregnancy might be purely a coincidence.

Dr. Swan stated that in three cases examination of the kidneys revealed glomerular sclerosis. Recently he had observed similar lesions in the kidneys of a baby whose mother had had a normal pregnancy. The precise significance of these lesions in relation to maternal rubella in pregnancy had therefore to await the examination of further material. Dr. Mervyn Evans had found congenital dental abnormalities in 23 of 34 babies whose mothers had suffered from German measles during pregnancy. One of the most prominent features was retardation of eruption. Dr. Swan stated that every effort should be made to acquaint the general public of the dangers associated with the contraction of German measles during pregnancy.

Lantern slides dealing with the pathology of the cataracts and of the kidneys were shown. In addition slides of the eyes of a three months' fœtus were shown. The mother had suffered from rubella when two months pregnant. Investigation of one eye disclosed considerable disorganization of structure associated with cataract, lack of closure of the fœtal fissure, microphthalmos and diminution in the number of blood vessels supplying the lens.

MISS IDA MANN (Oxford) sent a communication which referred to three aspects of the subject: (i) the electric phenomena and other protoplasmic activities that exist at different stages of development of multicellular organisms; (ii) the selective action of the placenta in its role as a barrier to the passage of certain substances noxious to the embryo; (iii) a comparison of the development of three structures damaged by maternal rubella, namely, the lens, the internal ear and the heart. Miss Mann concluded by expressing the opinion that the defects noticed first by Gregg were not a new complication of rubella, but a previously undetected result of it and possibly of other infections.

DR. F. V. SCHOLES (Melbourne) said that during the past thirty-five years, the first large epidemic of rubella had occurred in 1914, the next in 1923 and the last in 1937. The 1937 outbreak spread to all States, but had not died out in remote districts when war broke out in 1939. In that year, with the mass movements of young people from State to State and from country districts, the disease became epidemic, sweeping through the whole country, and waves had recurred until fairly recently. Between 1923 and 1937 there was no opportunity for anyone to contract rubella, and from 1937 to 1942 there was every opportunity, the vast majority of young adults and women of children-bearing age being susceptible. Dr. Scholes said that Dr. Gregg had amply proved his case, and the discovery was of great importance. It was clear that for protection of the fœtus, complete protection of the mother should be aimed at rather than attenuation of her illness, and therefore convalescent or pooled adult serum would need to be given immediately after exposure of the mother to the risk of infection. During an epidemic serum should be given as soon as possible after the beginning of pregnancy.

Cure of Naso-Lachrymal Obstruction.

DR. N. M. MACINDOE (Sydney) described a technique which consisted of dissecting up the sac from below and tying in a fine rubber tube. The lachrymal bone was then punctured with a round chisel and the tube passed into the hole on the end of a probe curved to three-quarters of a circle. Healing took nine or ten days and the tube was withdrawn from the nose. Nine cases were reported; eight were successful and one was a failure. In eight cases the new opening from sac to nose appeared to be permanent, as sepsis in the sac had cleared up and the epiphora was cured.

The Colour of the Human Iris.

DR. KEVIN O'DAY (Melbourne) pointed out that the variation in colour of the human iris was due to the amount of pigment present in the anterior layer of the stroma. Hence it was popssible, from a study of an iris fixed with formalin, to form an opinion as to the colour of the eye in life.

Advantages of Intracapsular Extraction for Cataract.

DR. E. V. WADDY POCKLEY (Sydney) read a paper dealing with what he considered to be the main advantages of intracapsular extraction for cataract and urged the wider adoption of this operation in this country. It was emphasized that in view of modern improvements in technique it need not be regarded as a procedure too hazardous for general and routine practice by ophthalmic surgeons. Experience at the Royal Prince Alfred Hospital over a lengthy period had confirmed this; no eye had been lost and no serious complication immediate or remote had been encountered which could be attributed to the intracapsular method of extraction. The main advantages were the following: (i) Operation might be undertaken at any stage, since the question of lens immaturity could be safely ignored. (ii) Less post-operative reaction occurred, owing to lack of lens remnants. (iii) The length of stay in hospital and the duration of convalescence were shortened. (iv) Aspiration of after-cataract was not needed. In conclusion, the view was expressed that in spite of repeated and strenuous opposition, intracapsular extraction, with its seductive claims, would return again and again until it achieved universal acceptance as the operation of choice.

Extraction of Cataract through a Keratome Incision.

DR. ARTHUR D'OMBRAIN (Newcastle) read a paper and described a simplified method of cataract extraction, using a keratome and scissors instead of a Graefe knife for the section and discarding the use of many of the auxiliary procedures in current surgical technique. The main advantages claimed were: accurate placement of the upper arc of the section exactly at the corneo-scleral limbus, a natural tissue junction; complete control of the eyeball during the keratome incision, the eye being held firmly between the grip of fixation forceps below and the steady push of the keratome above; absence of the need of multiple accessory procedures; absence of the need of a conjunctival flap, owing to exact apposition of the lips of the thin, corneo-scleral keratome incision with consequent early, firm union; ease of performance on the part of the surgeon and a less trying ordeal for the patient than a knife section with its attendant complex precautions.

A Simple Method of Lid Retraction and Eye Fixation in Cataract Extraction.

DR. F. GREGORY ROBERTS (Sydney) pointed out that the vast number of self-retaining specula and their modifications were indicative of the unsatisfactory nature of any of them. The method he described dispensed with the use of a self-retaining speculum, and included appropriate premedication and a preliminary facial and retrobulbar nerve block. The lids were separated by traction upwards on a switch inserted into the skin of the upper lid near the eyelash margin and opposite the 12 o'clock position of the cornea, the lower lid at the same time being held out of the way by a bent wire retractor. The eye, which had been immobilized by the retrobulbar nerve block, was fixed and maintained in the one position throughout the operation by very gentle traction upwards on a stitch through the superior rectus muscle, and by anchoring it in the correct position to the towels around the head. Thus the patient was 100% in the hands of the surgeon, and this added greatly to his confidence. The lids were separated without any pressure on the eye; the eye was completely immobilized and the procedure was as free from risk as any exposure of the eye could be.

Plastic Surgery.

MAJOR B. K. RANK, at an Australian general hospital, demonstrated several patients who had come under his care for surgical repair of facial injuries involving eyelids and orbit. He also showed cinematograph films illustrating the cases.

Penicillin.

MAJOR D. R. LESLIE dealt with the method of handling penicillin and with its use by intramuscular injection.

MAJOR MARK GARDNER (Melbourne) described successful treatment of conjunctivitis and dacryocystitis with drops containing 250 to 500 units of penicillin per cubic centimetre, and the cure of some early cases of keratitis by subconjunctival injection. He said that *herpes ophthalmicus* and cavernous sinus thrombosis had also been successfully treated.

The Treatment of Retinal Glioma by Radon Seeds.

DR. ARTHUR JOYCE (Melbourne) gave a short review of the recent literature before reporting the technique devised

for operating on a retinoblastoma, near the optic nerve on the nasal side. He said that retinoblastoma was the only glioma which formed metastases, and its method of spread followed definite channels. The main spread was along the optic nerve, but the tumour cells could reach the bones of the skull by way of the orbit or gain access to the subdural space through the nerve. The possibility of extension along the nerve was the main point to remember when an eye was being enucleated for glioma of the retina, and in this regard the opinion of various writers was given and different methods of dealing with the dangers were considered. Dr. Joyce said that the tumour in the case under discussion was in the only eye of a child of eleven months, the other eye having previously been removed for an inoperable glioma. The tumour was estimated by disk measurement to have an area of 3·0 millimetres by 2·25 millimetres; it was situated in the 10 o'clock meridian 2·25 millimetres to the nasal side of the optic disk, and it was raised one millimetre above the level of the surrounding retina. Measurements were made of an excised eye of a child of the same age. Owing to the fact that the nasal wall of the orbit was straight, it was impossible, in a position so far back, to suture the necessary four millimetre radon seed so that it would be rigidly fixed against the sclera. After the superior rectus, the internal rectus, and the superior oblique muscles had been reflected, the direction of the axis was marked with gentian violet, and along this line the required spot was accurately measured with calipers and marked with indian ink. A small tunnel was made in the episcleral tissue and the radon seed was introduced into it and the mouth of the tunnel closed with a suture. Ten months later the tumour area appeared as white scar tissue pigmented around the edges, and the surrounding area was not affected.

Contact Lenses.

DR. D. A. WILLIAMS (Sydney) gave some details of a technique which he had been using for the taking of impressions of the eyeball with atomized wax.

Crossed Cerebral Dominance.

DR. J. RINGLAND ANDERSON (Melbourne) read a paper prepared by Miss Celia Weigall and himself, in which the types of manual and ocular dominance and the means of testing them were discussed. It was stated that though they appeared frequently to be inherited, yet practice was necessary for their fixation. Crossed dominance was apt to be associated with nervous symptoms. Difficulty in learning to write, in playing games and in learning to fly had been experienced. These difficulties and the associated crossed dominance were probably evidence of an imperfectly integrated nervous system. Ocular dominance might interfere with the prescription of lenses and was certainly an obstacle in the training of children suffering from squint. Not only physical, but also emotional and intellectual gains, might follow the careful unilateralization in selected cases of ocular and manual dominance.

War and Ophthalmology.

DR. J. BRUCE HAMILTON (Hobart) reviewed his ophthalmic experiences in the Middle East (both army and civilian) and made the following eight suggestions for the immediate consideration of the Ophthalmological Society of Australia: (i) a vastly increased supply of ophthalmic sisters; (ii) an increase in special eye hospitals; (iii) the establishment of eye clinics for private practice in the larger capital cities; (iv) the establishment of mobile ophthalmic units for country districts; (v) ophthalmic training for discharged service medical officers; (vi) the stimulation of laboratory and clinical research by the society; (vii) the necessity for a new text-book in tropical ophthalmology; (viii) the obligation of Australian ophthalmology toward the peoples of the smaller colonies.

Annual Dinner.

The annual dinner, held at the Union Hall, University of Melbourne, was well attended and proved a most enjoyable function.

Annual General Meeting.

The annual general meeting dealt with routine matters, whilst particular attention was given to special problems. Matters discussed were the future of ophthalmology, visual hygiene and post-graduate education.

Dr. N. McA. Gregg (New South Wales) was elected President for the ensuing term.

It is proposed to hold the fifth annual general and scientific meeting at Sydney on October 3 to 6, 1945.

MEDICO-POLITICAL.

THE following are copies of letters that have passed between the General Secretary of the Federal Council of the British Medical Association in Australia and Commonwealth authorities regarding an application for authority to print for circulation a statement setting out the views of the Federal Council on the *Pharmaceutical Benefits Act*, 1944.

[COPY.]

FEDERAL COUNCIL OF THE BRITISH MEDICAL ASSOCIATION IN AUSTRALIA.

135 Macquarie Street,
Sydney,
19th October, 1944.

Comptroller General,
Department of Import Procurement,
52 William Street,
Sydney.

Dear Sir,

We wish to apply for an authority to print a statement setting out the views of the medical profession in regard to free pharmacy benefits for circulation.

It is estimated that the amount of paper required will be one and a half tons. Our printers are the Australasian Medical Publishing Company Limited, The Printing House, cnr. Seamer and Arundel Streets, Glebe,

Faithfully yours,
(Sgd.) J. G. HUNTER,
General Secretary.

COMMONWEALTH OF AUSTRALIA.

Division of Import Procurement,
Department of Trade and Customs,
52 William Street,
Sydney,
1st November, 1944.

National Security (Metal Foil and Paper) Regulations.

Dear Sir,

With reference to your letter of 19th October, 1944, requesting the issue of an authority to cover the use of one and a half ton of paper in the printing of a statement setting out the views of the medical profession in regard to free pharmacy benefits, it is desired that the following particulars be furnished to this office in order that the matter may be determined, viz:

(1) Copy of the proposed statement.
(2) Number to be printed, indicating number of pages, and size of page in each copy.
(3) Type of paper to be used; if other than newsprint state reasons why newsprint not suitable.
(4) Area of distribution and method to be adopted in distribution.

Yours faithfully,
(Sgd.) J. J. KENNEDY,
Comptroller General of Customs.

The General Secretary,
135 Macquarie Street,
Sydney, N.S.W.

FEDERAL COUNCIL OF THE BRITISH MEDICAL ASSOCIATION IN AUSTRALIA.

135 Macquarie Street,
Sydney,
23rd November, 1944.

Comptroller General of Customs,
Division of Import Procurement,
Department of Trade and Customs,
52 William Street,
Sydney.

National Security (Metal Foil and Paper) Regulations.

Dear Sir,

In reply to your communication of the 1st instant I now furnish as requested the following particulars:

1. A copy of the proposed statement.
2. (*a*) Number to be printed: 500,000. (*b*) Size and number of pages: single sheet, 11″ × 8½″.
3. Type of paper: newsprint.
4. Area of distribution and method to be adopted in distribution: throughout Australia per medium of members of the Association.

Faithfully yours,
(Sgd.) J. G. HUNTER,
General Secretary.

Enclosure.

COMMONWEALTH OF AUSTRALIA.

Division of Import Procurement,
Department of Trade and Customs,
52 William Street,
Sydney,
18th December, 1944.

National Security (Metal Foil and Paper) Regulations.

Dear Sir,

With reference to your letter, 1505, of 23rd November, 1944, concerning the printing of a statement on the Pharmaceutical Benefits Act, 1944, I wish to advise that before an authority to use paper for this purpose is issued this office needs to be assured that the statement is in conformity with the requirements of the State Publicity Censor.

Yours faithfully,
(Sgd.) J. J. KENNEDY,
Comptroller General of Customs.

The General Secretary,
Federal Council of the B.M.A. in Australia,
135 Macquarie Street,
Sydney, N.S.W.

FEDERAL COUNCIL OF THE BRITISH MEDICAL ASSOCIATION IN AUSTRALIA.

135 Macquarie Street,
Sydney,
22nd December, 1944.

Comptroller General of Customs,
Division of Import Procurement,
Department of Trade and Customs,
52 William Street,
Sydney.

National Security (Metal Foil and Paper) Regulations.

Dear Sir,

With further reference to the request of the Federal Council of the British Medical Association for authority to print a statement setting out the views of the medical profession in regard to free pharmacy benefits, I am now to advise you that the statement has been approved by the State Publicity Censor. (Censored copy attached, which I should be glad if you would return.)

It would be appreciated if further consideration could be given to the request of the Federal Council as set out in its letter of 19th October, 1944.

Faithfully yours,
(Sgd.) J. G. HUNTER,
General Secretary.

Enclosure.

FEDERAL COUNCIL OF THE BRITISH MEDICAL ASSOCIATION IN AUSTRALIA.

135 Macquarie Street,
Sydney,
17th January, 1945.

Comptroller General of Customs,
Division of Import Procurement,
Department of Trade and Customs,
52 William Street,
Sydney.

National Security (Metal Foil and Paper) Regulations.

Dear Sir,

With further reference to the request of the Federal Council of the British Medical Association in Australia for an authority to print a statement setting out the views of the medical profession in regard to free pharmacy benefits, *vide* my letters of the 19th October, 23rd November and 22nd December last, I am instructed by the Council to respectfully ask that a decision be made on the matter as soon as possible.

Faithfully yours,
(Sgd.) J. G. HUNTER,
General Secretary.

COMMONWEALTH OF AUSTRALIA.

Department of Trade and Customs,
Canberra, A.C.T.,
20th January, 1945.

Dear Sir,

Referring to my letter of 18th December, 1944, regarding your application for permission to use paper for the printing of a statement on the Pharmaceutical Benefits Act, 1944, for distribution by your Association, I would advise that further consideration of the application has been deferred pending the introduction of an amending bill during the next Session of Parliament.

Yours faithfully,
(Sgd.) J. J. KENNEDY,
Comptroller General of Customs.

The General Secretary,
Federal Council of the B.M.A. in Australia,
135-137 Macquarie Street,
Sydney, N.S.W.

Correspondence.

PUBLIC HEALTH ADMINISTRATION IN WESTERN AUSTRALIA.

SIR: The medical profession, no less than the public of Western Australia, owes you a debt of gratitude for your leading article of December 9 on "Public Health Administration in Western Australia".

It has become abundantly clear to all that an investigation is urgently required, but no statement has yet been made by the Government as to whether or not a royal commission will be granted. Dr. Park has left us, and I understand that before long your journal will be asked to publish an advertisement calling for applications for a new commissioner. It is essential, therefore, that some of the facts regarding the department be made known.

On December 20, 1944, the day on which Dr. Park departed from Western Australia, the Government "Roundsman" contributed an article to *The West Australian*, and *inter alia*, made the statement: "The Act is framed to give the Commissioner full authority." This statement is true, but it is not the wording of the act which is in dispute, but the administration of the department.

In practice, the Commissioner has not full authority, as can be demonstrated. To make it clear, it will be wise to point out that there is a Department of Public Health and a Medical Department, and that these two departments, while appearing to the public to be one, are administered in such a manner as to make it still a little doubtful as to whether they are separate departments or are combined.

I attach a copy of a memorandum which the Chief Secretary stated had been compiled as a result of a conference called to define the respective positions of the Commissioner of Public Health and the permanent head. In Section 2 of the Hospitals Branch, it will be noted that the following sentence appears: "The term has dropped into disuse together with the term 'Medical Department', since both have been concentrated under one Minister."

On October 17, 1944, I asked in the Legislative Council: "Has the Commissioner of Public Health administrative control of both the Health and Medical Departments?" I received the answer: "No. He is the professional head of the combined departments." A day or two later, I asked: "As the Commissioner is not in administrative control of either the Health or Medical Departments, who is?" Reply: "The Commissioner is responsible under the Minister for the administration of the *Health Act*. So far as the hospitals are concerned, the Commissioner is responsible for professional matters, or affords professional advice. The Under Secretary is the permanent head and is administratively responsible as such."

Thus, it becomes clear that the Commissioner is not in administrative control of the Health Department, but is responsible for the administration of the *Health Act*; and it will be noted that in the memorandum the wording is used, "the Commissioner is the 'administrator' of the *Health Act*".

It is exceedingly difficult to imagine that the Commissioner can administer the *Health Act* without having control of his department.

The Chief Secretary, in replying to my request for a royal commission, made confusion worse confounded when he said: "The whole Act centres round the Commissioner of Public Health, who has direct access to the Minister. There is no mention of the permanent head, yet Dr. Hislop suggests interference by the permanent head, although there is no legitimate basis for the suggestion."

Having been informed that the Commissioner does not control administratively the Health Department, and having been assured that there is no permanent head of that department, or if he exists apart from the Medical Depart-

ment he does not interfere, who does administer the Health Department? This is the complex administration which the Legislative Council asked should be investigated by a royal commission. It is this complexity which was responsible for Dr. Park's resignation, especially when the Minister in his reply to Dr. Park found himself unable to grant Dr. Park, as Commissioner, administrative control. In this, he stated: "I am certainly not in a position to give the 'definite undertaking' which you seek, that such a change will be effected." (Dr. Park had asked for a transference of administration to the Commissioner following the forthcoming early retirement of the present Under Secretary.)

Further points in the memorandum are worth consideration, because they show the conditions under which the Commissioner acts. I refer to this memorandum because I understand that it is the accepted basis for the new appointment. Number 4 reads: "To settle all medical appointments." It is difficult to understand what the word "settle" here means. Apparently the Commissioner has not the right to make an appointment subject to the Minister, and the word "settle" will undoubtedly lead to complications. It would appear that the Commissioner would only come into medical appointments were there any dispute.

Number 6 reads: "He should be consulted regarding all building proposals." This does not mean that his advice need be taken, and we have had many examples in which medical advice has in the past not been accepted, but the department has taken it that by having made its decisions known to a medical practitioner interested locally in the proposals, consultation with a professional man has occurred.

Number 8 of the memorandum will lead to difficulties, because if an Inspector of Hospitals is appointed, he will be under the direction of and able to report to the Commissioner; but again, the Commissioner's representations concerning hospitals will be purely advisory, because under Clause 7 the Commissioner himself can after an inspection only recommend concerning hospitals, but to whom, the clause fails to state.

It would be wise for all intending applicants for this post to be quite certain of their future status before applying. The Minister, in replying to Dr. Park, wrote: "It is hardly conceivable that when you applied for the position of Commissioner of Public Health you did not inform yourself of the system of control operating in this State."

May I make one final comment concerning salary? The salary offered is totally inadequate—£1,180, rising to £1,300, as against the suggestion of the National Health and Medical Research Council of £2,000—especially when it is realized that the Commissioner of Railways in this State receives £2,000 per year and the Director of Public Works £1,500.

If the salary of £1,180 is accepted as being fit for a Commissioner, what salary is likely to be offered for a medical man who will act as Inspector of Hospitals? This is a large State, with a relatively great number of hospitals for its population, and he who holds such a position would need to be a man of considerable experience in hospital administration.

I am of the opinion that the profession would still be wise to press for a royal commission as the only proper method of investigating the present administration in Western Australia.

Yours, etc.,

J. GORDON HISLOP.

Chennell House,
260, St. George's Terrace,
Perth.

January 11, 1945.

Memorandum.

A. Public Health Branch.

The Commissioner is the administrator of the *Health Act*, and of all associated health functions, such as school medical and dental work and infant health.

Under Public Service Regulations he is the subhead. He has direct access to the Minister, either personally or in writing.

The indications are that the clerical staff on the health side will need to be built up. The Under Secretary, by long association with public health, before the hospitals section was joined, has tended to assist the health administration. On his retirement it might be arranged that the Assistant Under Secretary shall specialize on health and also act as secretary to the Commissioner (a position provided by the *Health Act*).

B. Hospitals Branch (Hospitals and Medical Services and Homes).

1. Of the considerable volume of business, the bulk relates to finance, administration, legal matters, staffing.

2. The "P.M.O."[1] is the professional subhead. The term has dropped into disuse, together with the term "Medical Department", since both have been concentrated under one Minister. For instance, we appointed the "Commissioner of Public Health", not the "P.M.O." to the Medical Board.

3. The duties of the professional subhead are: to *visé* and approve all requisitions for drugs, instruments and appliances.

4. To settle, subject to the Minister, all medical appointments.

5. To deal with any professional matters which arise.

6. He should be consulted *re* all building proposals (other than of an obviously minor character).

7. He should visit and inspect hospitals as convenient and make recommendations as he thinks fit.

8. If a second doctor is appointed (a deputy commissioner) who acts as Inspector of Hospitals, the latter would be under the direction of and report to the Commissioner.

9. In all professional matters of this branch, as with the health side, the Commissioner would have direct access to the Minister.

SOCIOLOGICAL MEDICINE.

SIR: I was very interested in Dr. E. P. Dark's paper on "Sociological Medicine" as reported in THE MEDICAL JOURNAL OF AUSTRALIA of January 13, 1945. I am in full agreement with all that Dr. Dark says about the necessity of the medical profession being in the forefront in the fight to have the conditions of the people improved in every way.

However, one cannot agree that the figures Dr. Dark quotes as to the effect of poverty on the death rate at all justify the deductions he makes from them. He states that: "In families occupying four or more rooms in an English borough the death rate was 6·4 per 1,000. In families living in one room it was 39; that is, in the present state of knowledge, about six persons per 1,000 inevitably die of malignant disease, tuberculosis, diabetes, syphilis, heart disease and all other infections and chronic diseases, and 33 per 1,000 die of poverty." This deduction is quite illogical and could only apply if various other factors were the same in both groups. I would venture to suggest that if the ages in the two groups were compared there would be found a great discrepancy. It might be found, for instance, that a large percentage of those living in one room were old-aged pensioners and the like, the normal death rate of whom would be in the vicinity of 39.

The same argument applies to the next group dealing with wages. Here we have a group on a wage of 25s. to 35s. per week with a death rate of 25·96 per 1,000. Here again common sense would tell us that a large percentage of this group are pensioners or persons who from age or disease are doing part-time casual work.

Later on in the article Dr. Dark again makes a statement about the effect of the reduction in 10% of the food relief scale in England about 1932. Dr. Dark has stated repeatedly that this caused directly the deaths of 30,000 people per year in Great Britain for the next seven years during which it was in operation.

However, the official figures for the total number of deaths in Great Britain and the death rate for those seven years show no increase whatsoever over the general average for years before the application of this cut. In fact, 1929, a boom year, had a death rate and total number of deaths considerably greater than any one of the seven years during which Dr. Dark asserts 30,000 extra people died due entirely to this reduction of 10% in the food relief.

I myself must conclude that Dr. Dark's statement is incorrect and a slur on the British Medical Association which he blames for not pointing out that the 10% cut would cause 30,000 deaths per year.

Dr. Dark states that he is in favour of a salaried medical service. By inference he admits that it would be necessary for the patient to have choice of doctor and states that this could easily be arranged. At a meeting of the Blue Mountains District Medical Association, of which Dr. Dark is secretary, Dr. Dark voted for a motion that in the event of a salaried medical service being forced upon us, a doctor's hours should be limited to 44 hours per week. I would be pleased to hear how Dr. Dark proposes to see to it that the patients are going to arrange their illnesses, confinements, acute colics *et cetera* to occur during their chosen doctor's hours on duty.

[1] "P.M.O." refers to the appointment of Principal Medical Officer in the *Hospitals Fund Act*, an appointment which the Minister may make, but which has lapsed.

I believe that most doctors would for themselves prefer a salaried service with shorter hours, a suitable pension scheme *et cetera*. They oppose it because it would do away with a patient's choice of doctor for one thing and would not be in the interest of the patients themselves.

Dr. Dark has to my mind advanced no reasons why sociological medicine cannot be practised as efficiently and thoroughly under a democratic *régime* as under a socialistic one as he advocates. One wonders whether Dr. Dark's advocacy of the socialization of medicine does not depend on and indeed necessarily follow on the fact that he is in favour of a complete socialistic *régime* rather than because he thinks it is the best *régime* so far as the interests of the patients are concerned.

Yours, etc.,

A. C. TERNES.

Portland,
New South Wales,
January 17, 1945.

SIR: I am much impressed by your editorial on "Social Medicine", about which I have talked and written *ad nauseam*.

The great majority of pupils are taught geography, history, physics, chemistry *et cetera*, in all of which the facts are definite and the conclusions definite. But when they enter the world of living and thinking things, the facts are not always definite, and the conclusions are too often probabilities.

When sociology is a subject of universal instruction we shall get a better ordered and saner world. As the late Sir Horace Plunket put it: "The country man's politics are better than those of the city man, because he is dealing with social problems and knows that speeches and acts of parliament will not make his crops grow or his animals multiply."

The country man knows one job from A to Z. The city man too often knows only a portion, and sometimes only a very small portion, of one occupation. He sees life through a chink and thinks that by political action he can improve his own lot.

As it happens, medicine is a department of sociology to a great extent, and medical practitioners can, if they wish, exert a powerful influence on social affairs, whether it be playgrounds, housing, town planning or bush nursing. A visit to the new Bush Nursing hospitals and contact with the committees is an education. But, of course, the knowledge of medical practitioners will only be valuable if they take some interest in the multitudinous problems around them. I myself cannot imagine an ordered and stable society which does not understand the mechanism and functions of its individual members, and realize the hard and fast conclusions in its daily work and the harm it does.

Yours, etc.,

JAMES W. BARRETT.

105, Collins Street,
Melbourne,
January 19, 1945.

SIR: May I make a few comments on Dr. Dark's article in the journal of the thirteenth instant?

It must be beatific to have derived such a simple formula for the solution of the world's problems as has Dr. Dark. It alarms me a little to read his exultant reference to opinions that a social revolution is in progress, considering the amount of mortality and suffering caused by the religious revolution of the Middle Ages and by the French and Russian political revolutions, but I retain the hope that the evolutionary methods, of which Britain has been the able exponent, will still prevail with us.

I believe with Dr. Dark that "facts should be followed relentlessly to their ultimate causes". Yet his reasoning in dealing with statistics seems to me to smack too much of sentiment and too little of science. But perhaps I am archaic in thinking that heredity is still a factor in development. He concludes that the sole cause of the higher infantile mortality among the low income group than among the high income group is poverty. But it is conceivable that a man's income depends, *inter alia*, on his inherited physical constitution and intelligence (generally speaking, the poorer the constitution and intelligence, the lower the income), and the chance of survival of infants will also depend on the constitution inherited and the intelligence shown by the parents in their upbringing. Even in environment there are other factors than poverty.

Dr. Dark's death rates in relation to income call for similar criticism. He deals only with the rate for all age groups combined, assuming that all the income groups have the same or, at least, a similar age distribution, a condition that cannot be taken for granted. Again, he ignores the probability that a poor constitution with a lower expectation of health and life may be the cause rather than the result of the low income. The same applies to his remarks on anthropometry, and surely the development of intelligence depends on many factors. His conclusions cannot be accepted without a more relentless search for ultimate causes.

We want to see work and good working and living conditions provided for all, and payment made according to the work done; to see a sound education given to all in so far as they are able and willing to absorb it on how to live and how to think and how to act fairly to the State. During the last depression in this district there were men who preferred to live in town, where, from various sources, they received shelter, food and clothing and the enjoyment of pictures twice a week, rather than go to the country and work on a farm. Education will have to be deep and broad.

It is a reflection on the validity of Dr. Dark's argument that the privileged classes are hostile to the education of the masses when he has recourse to a speech made in 1807. This was the period of the Napoleonic wars when the comparatively recent wholesale murders of the privileged classes by the masses in France had caused much general distrust and bitterness. What would Dr. Dark retort if, in reply to his statistics, similar statistics in support of the rich and well-housed were quoted for France and Russia covering the period of their respective revolutions? Yet in 1807, even in Tsarist Russia, according to Tolstoi, there were privileged people who were enthusiastic about educating the masses.

Does Dr. Dark really mean that reformatories for child delinquents are incubators for mature criminals because nearly half the prisoners in Goulburn gaol had passed through the Gosford Boys' Home? Surely this censure is unjustified by the evidence adduced. Dr. Dark states, but does not prove, that "most [a vague word here] criminals are made by their environment". However, I suggest that those prisoners were probably in Goulburn gaol in spite of the reformatory because of inherited criminal attributes.

Thirty or more years ago, when slum-dwellers were moved to a new building of flats in Glasgow, in a short time the building was beginning to be slum-like and a caretaker had to be installed.

If the slums produce youths who can afford to devote their time to "pubs, pitchers and ponies", as Dr. Hewitt stated, then they can't be so poor. Yet I warrant these people think they get more out of life than such as I, whom they would jeer at as a bookworm.

Having inherited the Scottish tradition, I am all for intensive research and effective education, unbiased by politics.

Yours, etc.,

J. BROWN.

Toowoomba,
Queensland,
January 20, 1945.

Obituary.

LIONEL WILFRED BOND.

WE are indebted to Professor W. S. Dawson for the following tribute to the late Dr. Lionel Wilfred Bond.

Lionel Wilfred ("Lyle") Bond, whose death occurred recently, was educated at King's College, Goulburn, after which he proceeded to Sydney University (Saint Andrew's College). After graduating M.B., Ch.M. in 1903 he held resident appointments at Sydney Hospital and at Toowoomba and Goodna Mental Hospitals. He enlisted early in 1915 and became registrar at the Second Australian General Hospital. He was then posted to the Eighth Field Ambulance and to the Fifty-Ninth Battalion, with which he served until he was wounded at Bullecourt, where he was awarded the D.S.O. The citation stated: "His total disregard of danger under a terrific hail of gas shells, H.E. and shrapnel fire, gained him the confidence of all ranks, and greatly assisted the evacuation of the wounded. Later, although wounded and partly gassed, he refused to leave his post, and his bravery and devotion saved a very critical situation." After recovery from his injuries he was posted to the Ninth Field Ambulance. After the Armistice he was in command of the Twenty-First Australian Army Hospital and of the Fourth Australian General Hospital. He received two mentions in dispatches and was demobilized with the

rank of lieutenant-colonel. He was for a time medical superintendent of the Prince of Wales Hospital, Randwick. He then devoted his time to psychiatry and was appointed honorary psychiatrist and neurologist at Lewisham Hospital and assistant physician to the Department of Psychiatry, Royal Prince Alfred Hospital, and later honorary physician, becoming consultant on reaching the age of limit in 1939. He was a Foundation Fellow of the Royal Australasian College of Physicians.

Ill health over a number of years no doubt accentuated a retiring disposition and he took little part in medical activities outside his practice and hospital appointments. He was a loyal and trusted colleague whose opinon was valued on repatriation tribunals and military medical boards, and his kindly manner, courtesy and understanding were appreciated by his patients, by whom he will be greatly missed.

JAMES FREDERICK WATSON.

WE regret to announce the death of Dr. James Frederick Watson, which occurred on January 22, 1945, at Vaucluse, New South Wales.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 14, of January 18, 1945.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Medical Branch.

The following Temporary Squadron Leaders are granted the acting rank of Wing Commander whilst occupying Wing Commander posts with effect from 1st December, 1944: F. F. Ellis (261200), D. T. Shortridge (281472).

The appointment of Temporary Flight Lieutenant J. J. Rice (282908) is terminated at his own request with effect from 28th November, 1944.

The appointment of Temporary Squadron Leader A. H. Penington (251211) is terminated on medical grounds with effect from 27th November, 1944.

Reserve: Medical Branch.

Temporary Squadron Leader P. E. Breheny (251272) is transferred from the Active List with effect from 20th November, 1944.—(Ex. Min. No. 22—Approved 17th January, 1945.)

Nominations and Elections.

THE undermentioned have applied for election as members of the South Australian Branch of the British Medical Association:

Brookman, Benjamin Edward, M.B., B.S., 1944 (Univ. Adelaide), Royal Adelaide Hospital, Adelaide.

Dinning, Trevor Alfred Ridley, M.B., B.S., 1944 (Univ. Adelaide), 40, Eaton Street, Malvern, South Australia.

Hunter, Ronald, M.B., B.S., 1944 (Univ. Adelaide), 26, The Crescent, Blair Athol.

Flecker, Patrick Oscar, M.B., B.S., 1944 (Univ. Adelaide), 3, Dickens Street, Saint Kilda, S.3, Victoria.

Colton, Robert Stirling, M.B., B.S., 1944 (Univ. Adelaide), 79, Moseley Street, Glenelg.

Hetzel, Basil Stuart, M.B., B.S., 1944 (Univ. Adelaide), 3, Newcastle Street, Heathpool.

Linn, Howard Wadmore, M.B., B.S., 1944 (Univ. Adelaide), 12, Balham Avenue, Millswood.

The undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Brenner, Maksymiljan, M.B., B.S., 1942 (Univ. Queensland), 465, Oxford Street, Paddington.

Davies, Geoffrey Wyndham, M.B., 1939 (Univ. Sydney), 55, Wright's Road, Drummoyne.

Paul, Alister Bulkeley, M.B., B.S., 1942 (Univ. Sydney), Captain A. B. Paul, 1 Australian Armoured Regiment, A.I.F., Australia.

Diary for the Month.

FEB. 2.—Queensland Branch, B.M.A.: Branch Meeting.
FEB. 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
FEB. 6.—New South Wales Branch, B.M.A.: Special Groups Committee.
FEB. 7.—Victorian Branch, B.M.A.: Branch Meeting.
FEB. 7.—Western Australian Branch, B.M.A.: Council Meeting.
FEB. 9.—Queensland Branch, B.M.A.: Council Meeting.
FEB. 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
FEB. 19.—Victorian Branch, B.M.A.: Hospital Subcommittee.
FEB. 19.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.
FEB. 20.—New South Wales Branch, B.M.A.: Medical Politics Committee.
FEB. 20.—Victorian Branch, B.M.A.: Organization Subcommittee.
FEB. 22.—Victorian Branch, B.M.A.: Executive Meeting.
FEB. 23.—Queensland Branch, B.M.A.: Council Meeting.
FEB. 27.—New South Wales Branch, B.M.A.: Ethics Committee.
FEB. 28.—Victorian Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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